BENIGN ESOPHAGEAL DISEASES

Richard C. Golding, M.D., M.P.H.

Introduction

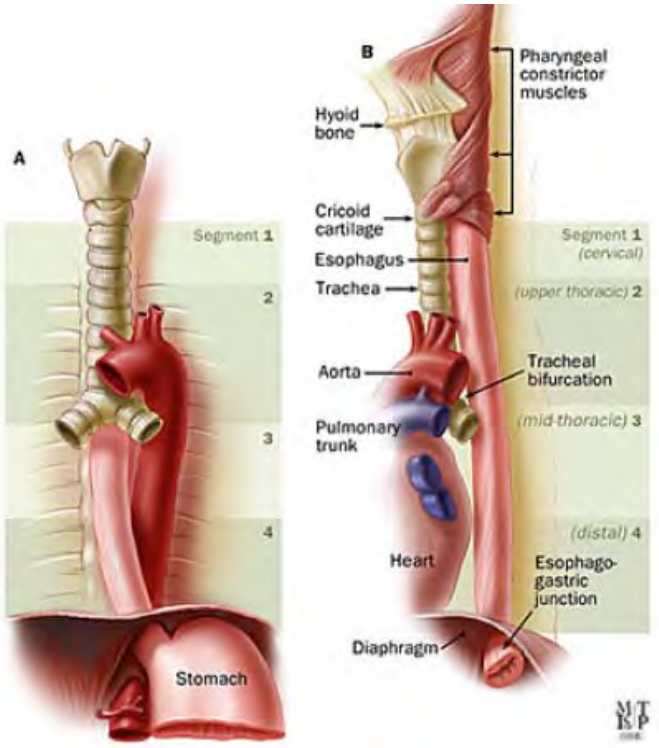
Outline

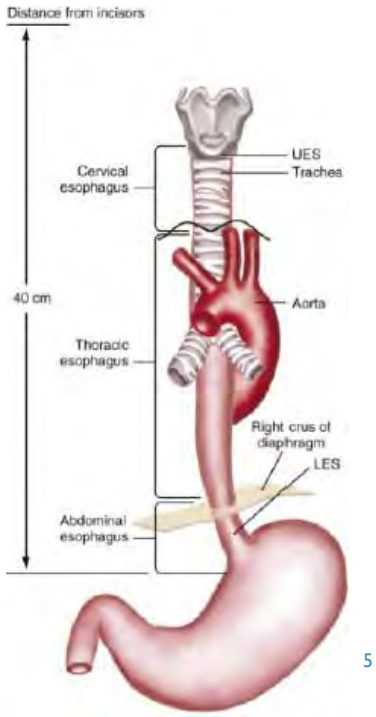
- Esophageal Anatomy
- Clinical Presentation of Benign Esophageal Diseases
- Structural Lesions of the Esophagus
- Esophagitis
- Gastroesophageal Reflux Disease (GERD)
- Motility Disorders of the Esophagus
- Esophageal Symptoms in Patients After Bariatric Surgery

ESOPHAGEAL ANATOMY

Esophagus

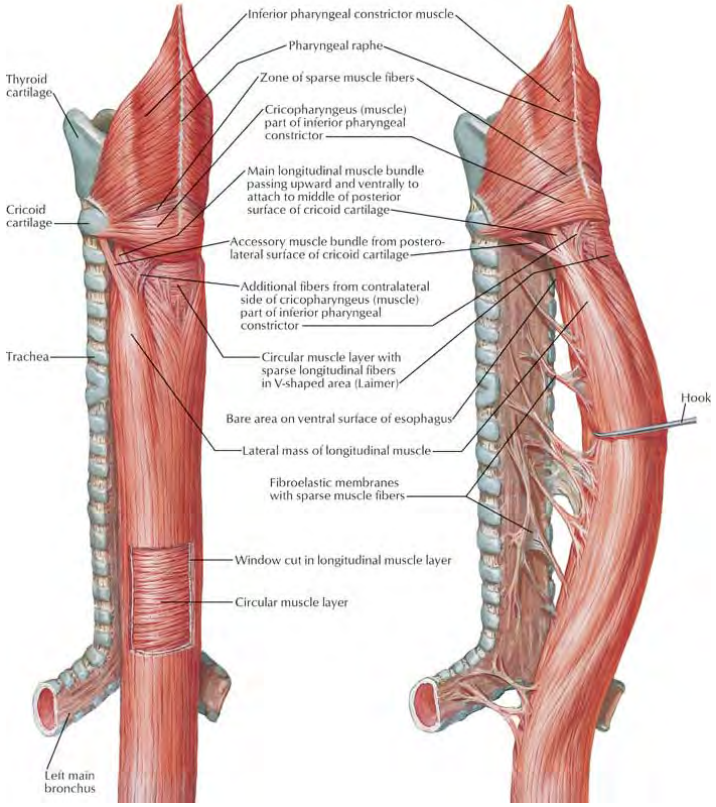
- Muscular tube whose primary function is to deliver swallowed material from the mouth to the stomach
- 25cm in length
  - Measured from its origin in the neck just below the cricoid cartilage





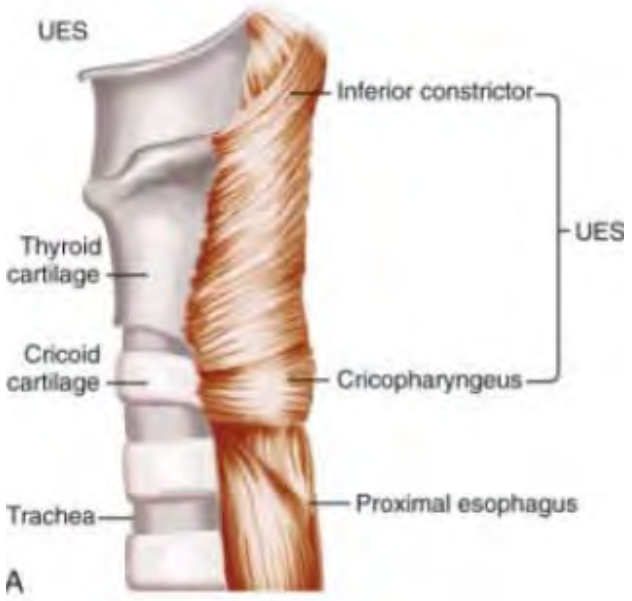
Muscles of Esophagus

- Outer layer of longitudinal muscle
  - Contraction causes the esophagus to shorten
- Inner layer of circular muscle
  - Responsible for squeezing motion that affects peristalsis and closure of esophageal sphincters



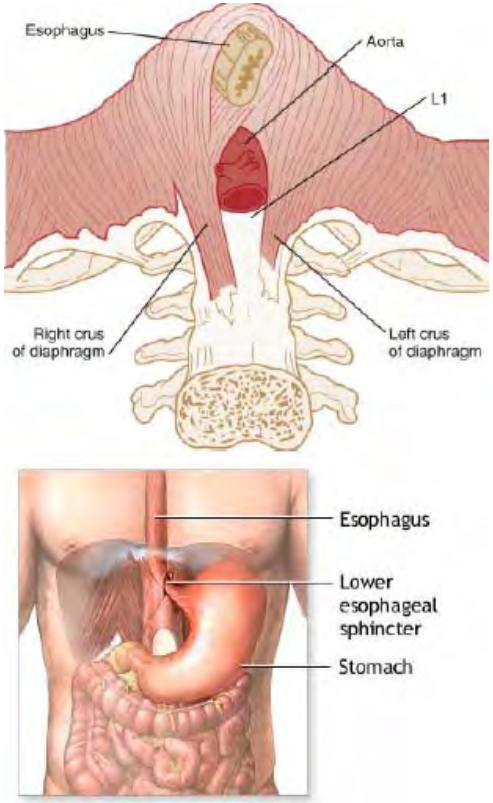
Upper Esophageal Sphincter (UES)

- Separates the pharynx from the esophagus
- 3cm in length
- Three skeletal muscle groups
  - Inferior constrictor
  - Cricopharyngeus
  - Proximal esophagus



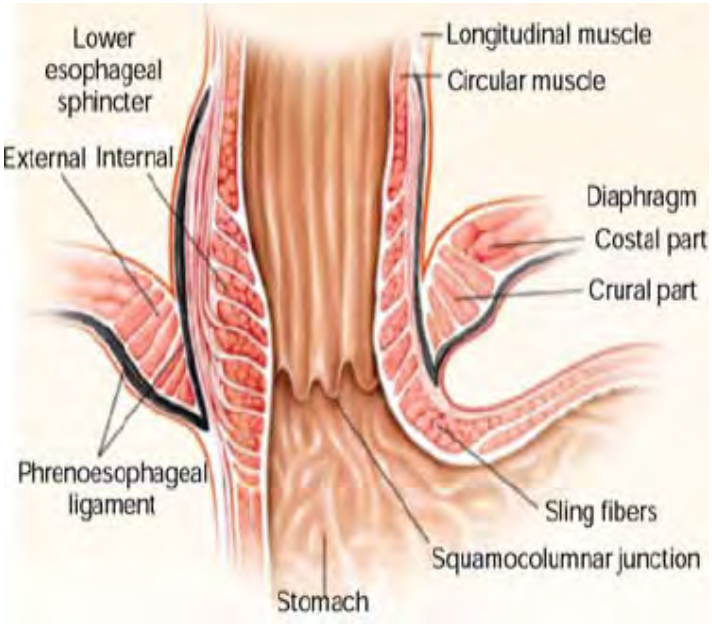
Diaphragmatic Hiatus

- The esophagus passes from the chest into the abdomen through the diaphragmatic hiatus
- Approximately 2cm of the distal esophagus normally lies within the abdomen



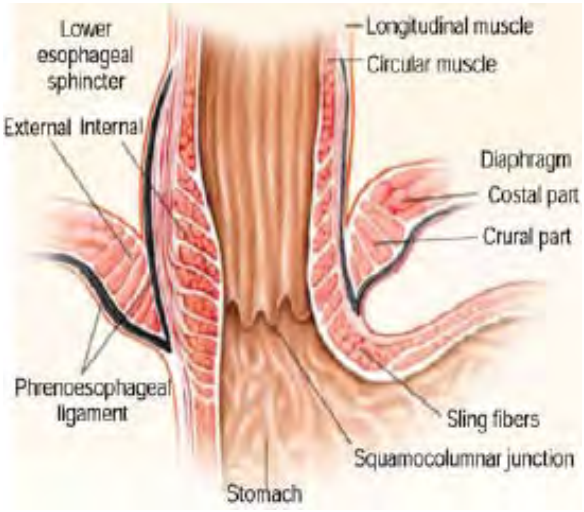
Lower Esophageal Sphincter (LES)

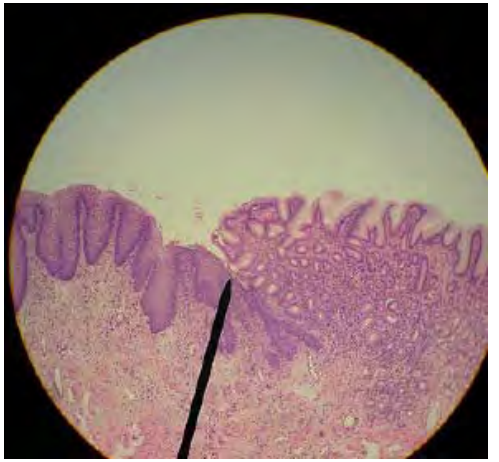
- 3cm in length
- External
  - Skeletal muscle of the crural diaphragm
- Internal
  - Smooth muscle of the distal esophagus



Z Line

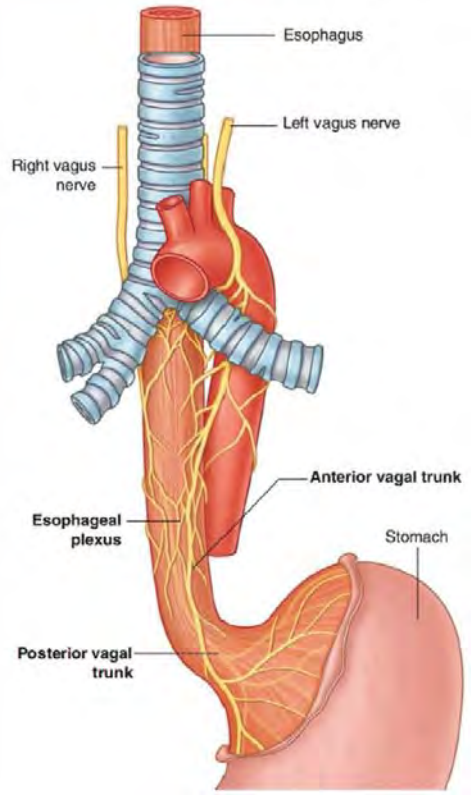
- Junction between the esophageal squamous epithelium and gastric-type columnar epithelium
- 1cm below the sphincter's proximal border





Innervation of Esophagus

- The esophagus, at baseline, is in a contractile state
- Peristalsis
  - Net result of the coordinated relaxation and contraction mediated by the inhibitory and excitatory myenteric plexus neurons along the length of the esophagus
- UES
  - Striated muscle
  - Depends on tonic excitation to maintain contractility
  - If innervation lost = flaccid
- LES
  - Smooth muscle
  - Inhibitory and excitatory effector neurons in myenteric plexus
- Proximal esophagus is subject to diseases that affect striated muscle and its CNS innervation
  - Polymyositis
  - Myasthenia gravis
- Distal esophagus is susceptible to diseases of smooth muscle and enteric neurons
  - Scleroderma
  - Achalasia



CLINICAL PRESENTATION OF ESOPHAGEAL DISEASES

Symptoms of Benign Esophageal Diseases

- Dysphagia
  - Oropharyngeal
  - Esophageal
- Heartburn
- Chest pain
- Belching
- Extra-esophageal symptoms (primarily associated with GERD, often presenting to ENT/Pulm physicians)
  - Globus
  - Halitosis
  - Laryngopharyngeal reflux (LPR)
  - Asthma
  - Cough
  - Sore Throat
  - Hoarseness

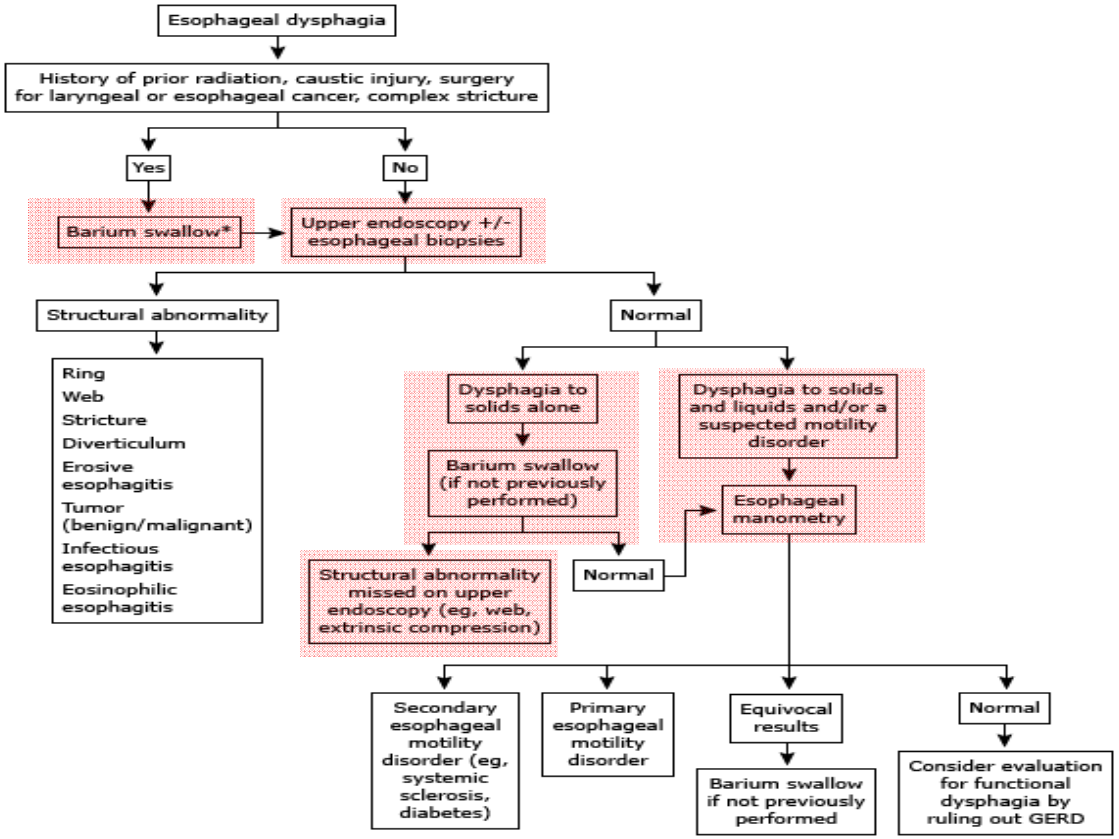
Evaluation Of Benign Esophageal Diseases

- Barium esophagram
  - Cervical and thoracic with barium tablet (13mm)
  - Symptomatic usually with lumen less than 13 mm
- EGD
- Modified barium swallow (MBS)
- Esophageal manometry
- pH studies

Diagnostic Studies

- Useful for evaluating esophageal disease
- Barium esophagram
  - With cervical and thoracic views
  - With 13.5mm barium table
- Modified Barium Swallow (MBS)
  - Done with speech therapy and radiology
  - Most useful for evaluation of oropharyngeal dysphagia
- EGD/biopsies
- Esophageal manometry
  - High Resolution Esophageal Manometry (HREM)
- Multi-channel Intraluminal Impedance (MII)
  - MII EM
  - MII pH
- Laryngoscopy
- pH studies
  - 24 hour pH
  - BRAVO

Evaluation of Esophageal Dysphagia

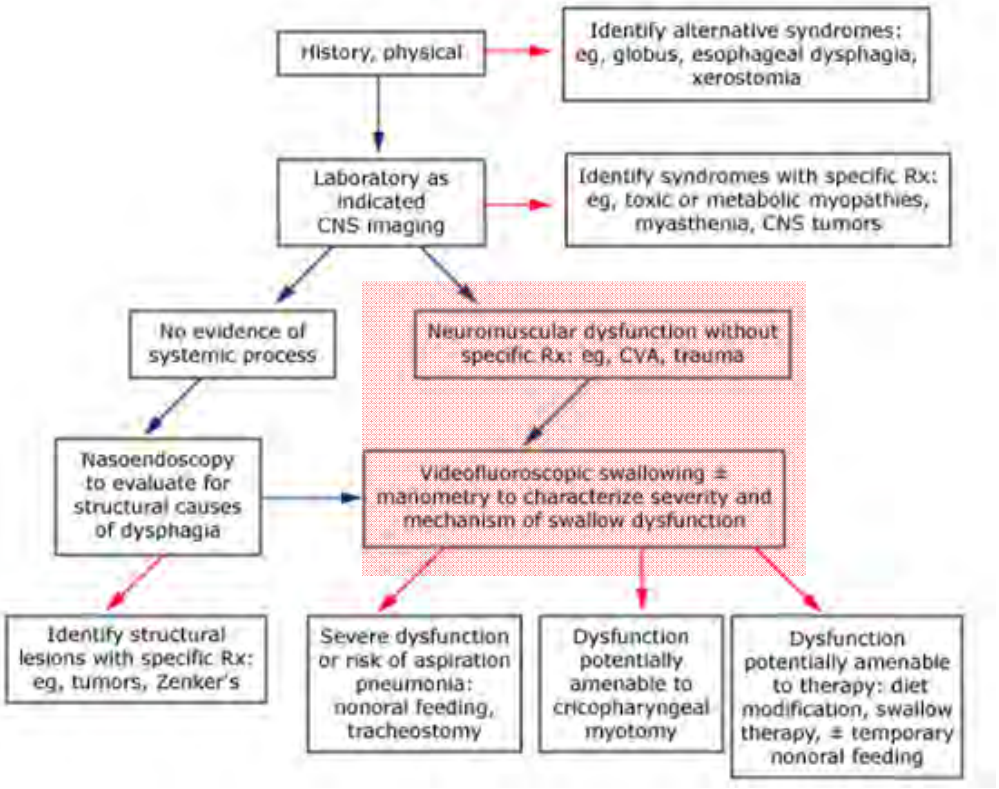


Causes Of Oropharyngeal Dysphagia

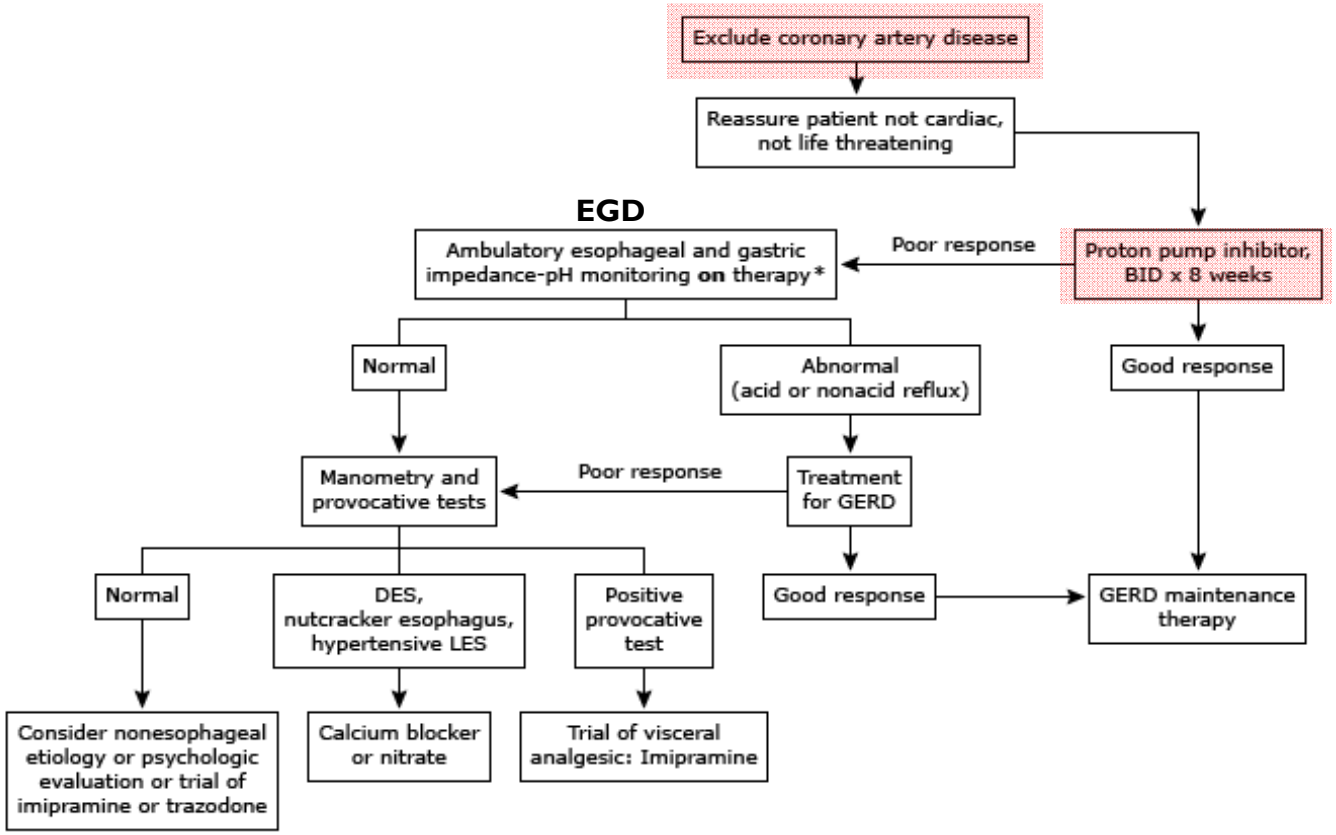
Iatrogenic
Medication side effects (chemotherapy, neuroleptics, etc)
Postsurgical muscular or neurogenic
Radiation
Corrosive (pill injury, intentional)
Infectious
Mucositis (herpes, cytomegalovirus, Candida, etc)
Diptheria
Botulism
Lyme disease
Syphilis
Metabolic
Amyloidosis
Cushing's syndrome
Thyrotoxicosis
Wilson disease
Myopathic
Connective tissue disease (overlap syndrome)
Dermatomyositis
Myasthenia gravis
Myotonic dystrophy
Oculopharyngeal dystrophy
Polymyositis
Sarcoidosis
Paraneoplastic syndromes

Neurological
Brainstem tumors
Head trauma
Stroke
Cerebral palsy
Guillain-Barré syndrome
Huntington disease
Multiple sclerosis
Polio
Postpolio syndrome
Tardive dyskinesia
Metabolic encephalopathies
Amyotrophic lateral sclerosis
Parkinson disease
Dementia
Structural
Cricopharyngeal bar
Zenker's diverticulum
Cervical webs
Oropharyngeal tumors
Osteophytes and skeletal abnormalities
Congenital (cleft palate, diverticula, pouches, etc)

Evaluation/Management Of Oropharyngeal Dysphagia



Evaluation Of Unexplained Chest Pain



STRUCTURAL LESIONS OF THE ESOPHAGUS

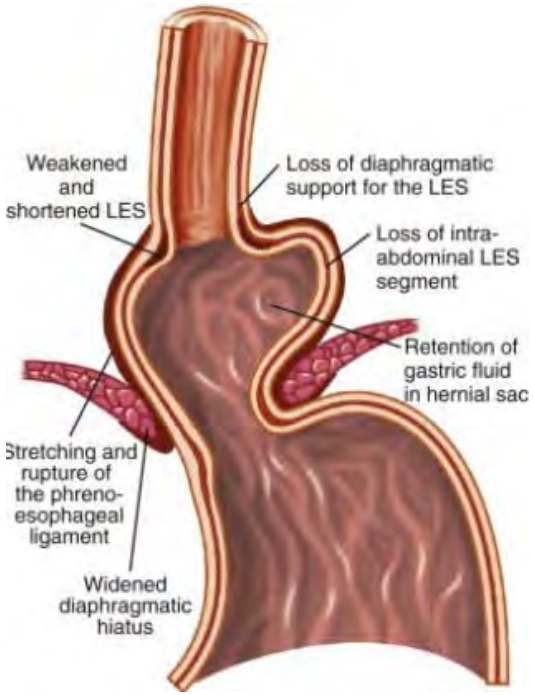
Structural Lesions

- Associated with anatomic narrowing
- Usually presenting with dysphagia
- When luminal diameter <13 mm
- Hernias
  - Hiatal
  - Paraesophageal
- Rings
- Webs
- Food impaction
- Foreign bodies
- Diverticulum

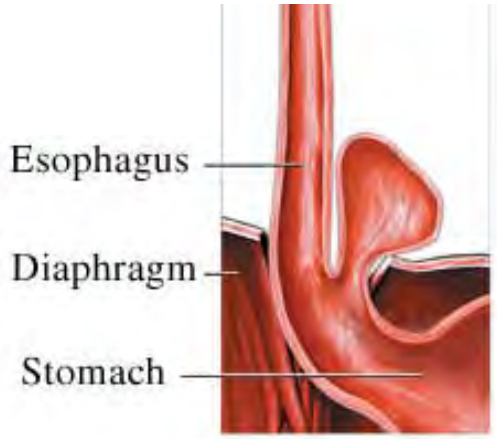
Hiatal Hernias

- Sliding hiatal hernia
  - Displacement of the internal LES from the crural diaphragm into the chest
- Paraesophageal hiatal hernia
  - Part of the stomach protrudes into the chest next to the esophagus

Sliding Hernia



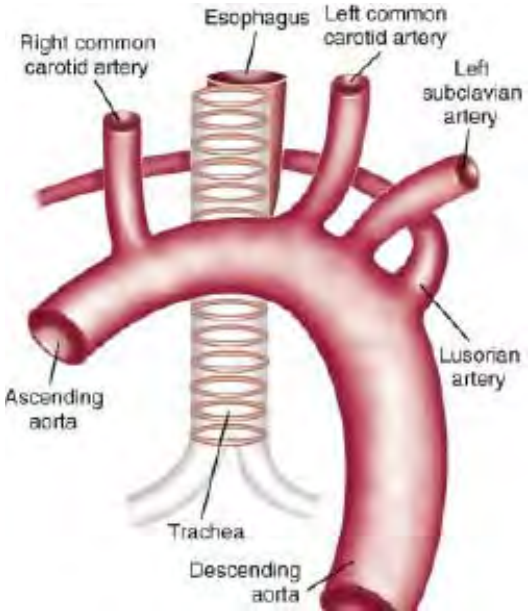
Paraesophageal Hernia



Vascular Anomalies

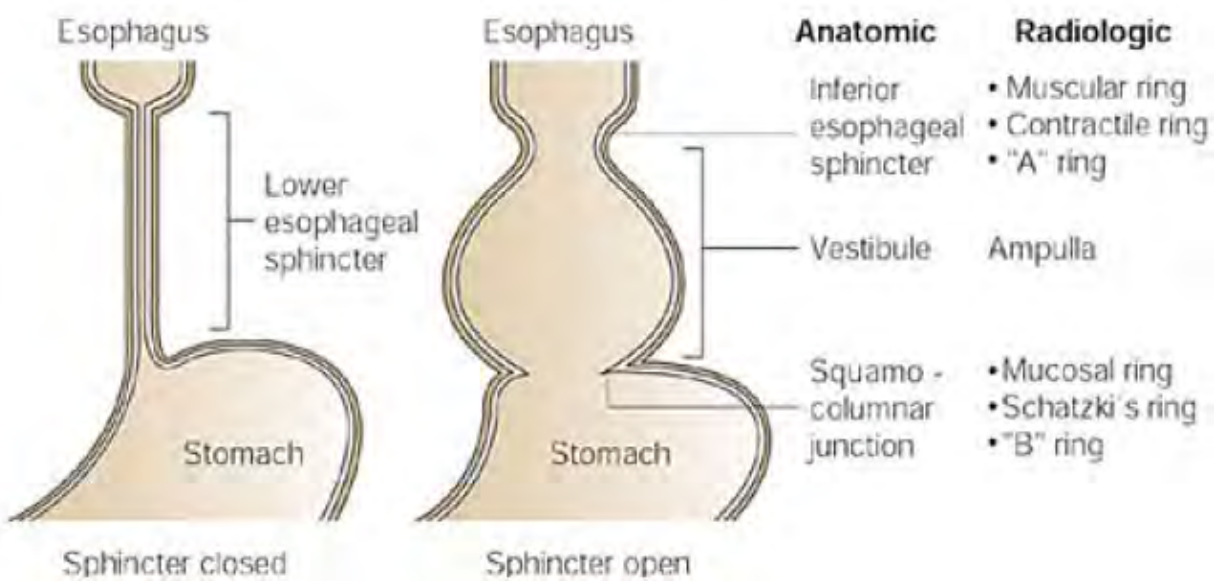
- Vascular anomalies
  - Intrathoracic vascular anomalies are present in 2-3% of the population
  - Only rarely do they produce symptoms of esophageal obstruction
  - Dysphagia Lusoria ("trick of nature")
    - Impingement of aberrant right subclavian artery on proximal esophagus

Dysphagia Lusoria



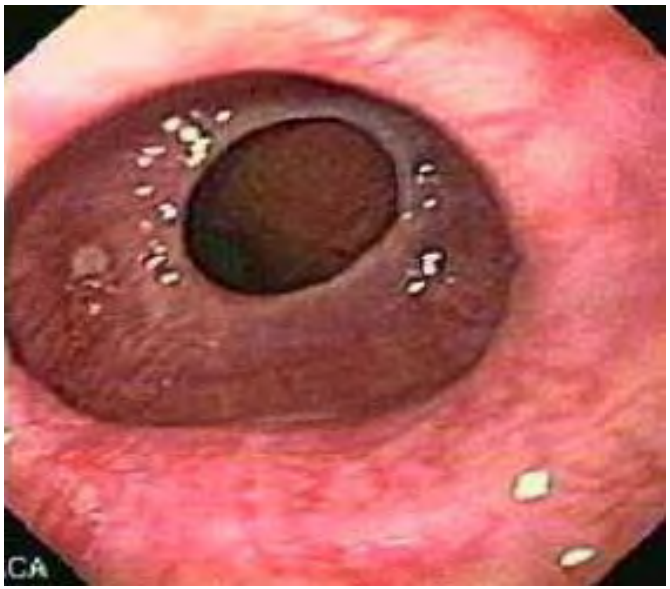
Distal Esophageal Rings

- Type A ring
- Type B ring
  - Known as Schatzki ring or Kramer-Inglefinger ring



Type A Ring

- Broad band of hypertrophied muscle that constricts the lumen
- Corresponds to the upper end of the LES
- Rare
- Generally asymptomatic
- Treatment if symptomatic
  - 50-French mercury-weighted esophageal dilator
  - Botox

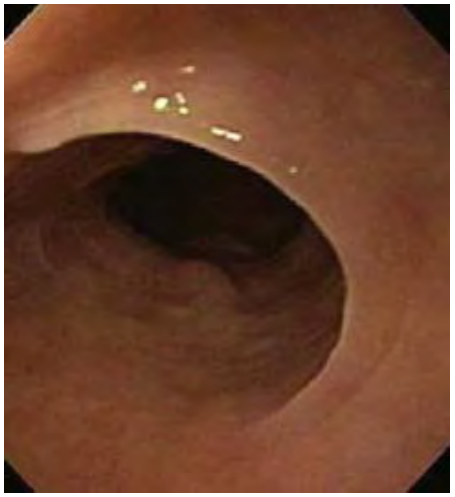


Type B Ring

- Schatzki's ring
- 4% of endoscopies
- Thin membrane at squamocolumnar junction
- Composed of only mucosa and submucosa
- Congenital or acquired
- Most asymptomatic, but can be associated with GERD
- Symptomatic
  - Diameter <13mm
  - Passage of single (≥50-French) bougie or (18-20mm) balloon dilator

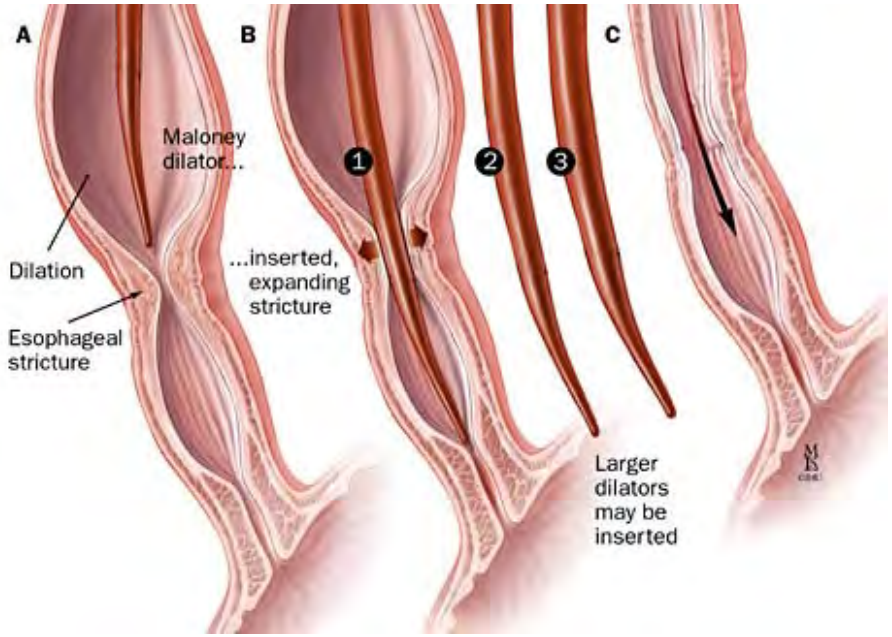
Esophageal Webs

- Common in the cervical esophagus
- Developmental anomalies
  - Thin horizontal membranes of stratified squamous epithelium
- Rarely encircle the lumen
- Best demonstrated on an esophagogram with the lateral view
- Cause dysphagia for solids when symptomatic
- Respond well to esophageal bougienage with mercury-weighted dilators

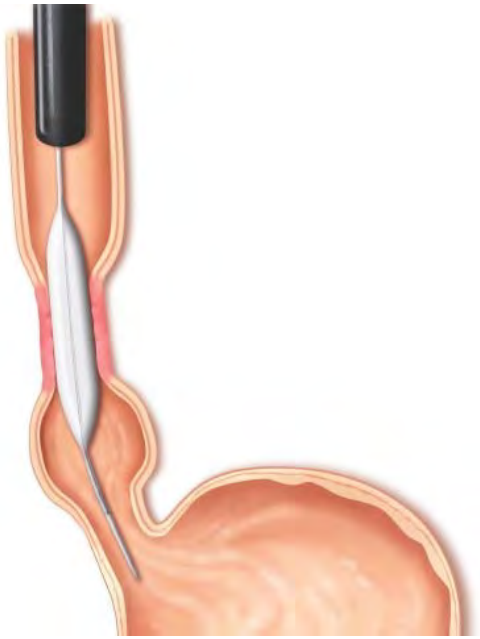


Esophageal Dilation

Maloney or Savary (guidewire) Dilation



Balloon Dilation



Plummer-Vinson (Paterson-Kelly) Syndrome

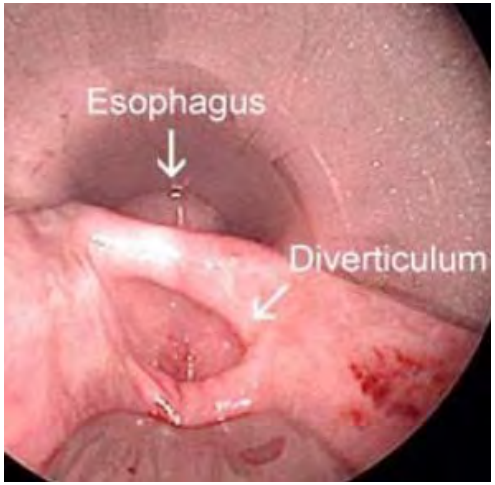
- Characterized by:
  - Cervical esophageal webs
  - Dysphagia
  - Iron deficiency anemia
- Primarily in women
- Associated with celiac disease
- Increased risk for squamous carcinoma of the pharynx and esophagus
- Correction of iron deficiency may result in resolution of the dysphagia and disappearance of the web

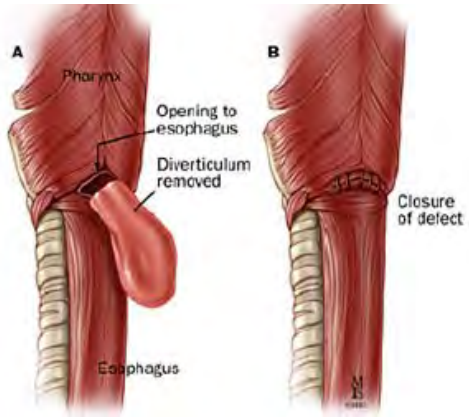
Zenker's Diverticulum

- A sac formed by the herniation of mucosa and submucosa of the hypopharynx through Killian's dehiscence
- Killian's area
  - An area of weakness in posterior wall of hypopharynx just above the cricopharyngeus muscle
- May result from poor distensibility of the UES muscles caused by fibrosis (wear and tear of swallowing over many decades)
- Over time, high pressure forces more of the mucosa to herniate through Killian's dehiscence and diverticulum enlarges
- Symptoms
  - Gurgling in the neck
  - Regurgitation of undigested food
  - Halitosis
  - Visible lump on side of the neck
  - Large diverticula can push on the esophagus causing dysphagia

Zenker's Diverticulum

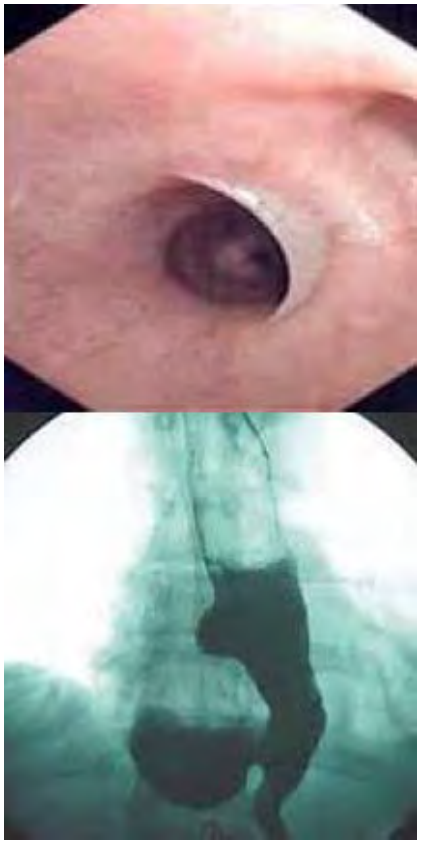
- Diagnostic studies
  - EGD
  - Barrium swallow
- Treatment
  - Surgical or endoscopic
  - Diverticulectomy
  - Should also have cricopharyngeal myotomy to prevent recurrence





Epiphrenic Diverticulum

- Arises from distal esophagus
- Commonly associated with underlying spastic esophageal motility disorder
- Can increase in size resulting in food retention and regurgitation
- Treatment
  - Surgical
    - Diverticulectomy
  - Treatment of underlying motility disorder



Case

- 34 yo male reports difficulty swallowing solids and a sense of fullness in his throat
- On a recent date, he chocked on a piece of steak and his girlfriend was frightened to see a fleshy tube snap out of his mouth and then snap back
- She ran away in horror and never came back

Case

Giant Fibrovascular Polyp

- Variety of lesions including fibromas, fibrolipomas, myomas, and lipomas
- Contain a mixture of fibrous, vascular, and adipose tissue covered by squamous epithelium
- Usually located in upper third of the esophagus
- 75% in men
- Age 50s-60s
- Up to 20cm

Fibrovascular Polyps

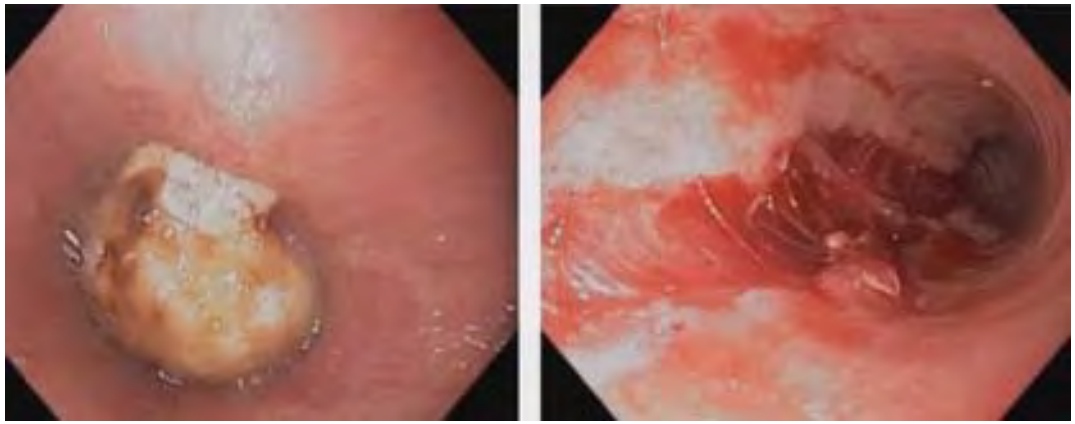
- Symptoms
  - Most asymptomatic
  - Case reports of large lesions causing asphyxiation
  - Dysphagia
- Treatment
  - Snare polypectomy
  - EUS should be performed before excision to rule out the presence of a large vessel feeding the stalk
  - Surgical resection if large feeding vessel is present or technically unable to remove endoscopically

Fibrovascular Polyp

Case: Food Impaction

- 24 yo male with 4-5 years of solid food dysphagia, seasonal allergies, and asthma, presents with food impaction
- Endoscopic devices for removing food impaction
  - Snares
  - Forceps
  - Nets
  - Graspers
  - Baskets

Food Impaction



Timing Of Endoscopy For Ingested Foreign Bodies

Emergent endoscopy
- Patients with esophageal obstruction (ie, unable to manage secretions)
- Disk batteries in the esophagus
- Sharp-pointed objects in the esophagus

Urgent endoscopy **(within 24 hours)**
- Esophageal foreign objects that are not sharp-pointed
- Esophageal food impaction in patients without complete obstruction
- Sharp-pointed objects in the stomach or duodenum
- Objects >6 cm in length at or above the proximal duodenum
- Magnets within endoscopic reach

Nonurgent endoscopy
- Coins in the esophagus may be observed for 12-24 hours before endoscopic removal in an asymptomatic patient
- Objects in the stomach with diameter >2.5 cm
- Disk batteries and cylindrical batteries that are in the stomach of patients without signs of GI injury may be observed for as long as 48 hours. Batteries remaining in the stomach longer than 48 hours should be removed.

Decisions Regarding Foreign Body Management

- Airway protection
  - Intubation may be required for upper esophageal obstructions
- Overtube
- Endoscopic hood for sharp objects
- Radiologic localization prior to extraction
- Thoracic surgery or ENT referral for foreign bodies not amenable to endoscopic removal

ESOPHAGITIS

CAUSES OF ESOPHAGITIS

- GERD
- Pills/medication related
- Caustic ingestion
  - Acids
  - Alkalis
  - Causing severe esophagitis with long strictures
- Radiation
  - Usually mediastinal
- Infections
  - CMV
  - Herpes simplex
  - HIV
  - Candida/fungal
  - Graft vs. host disease – BMT patients
- Eosinophilic esophagitis (EoE)
- Pemphigus

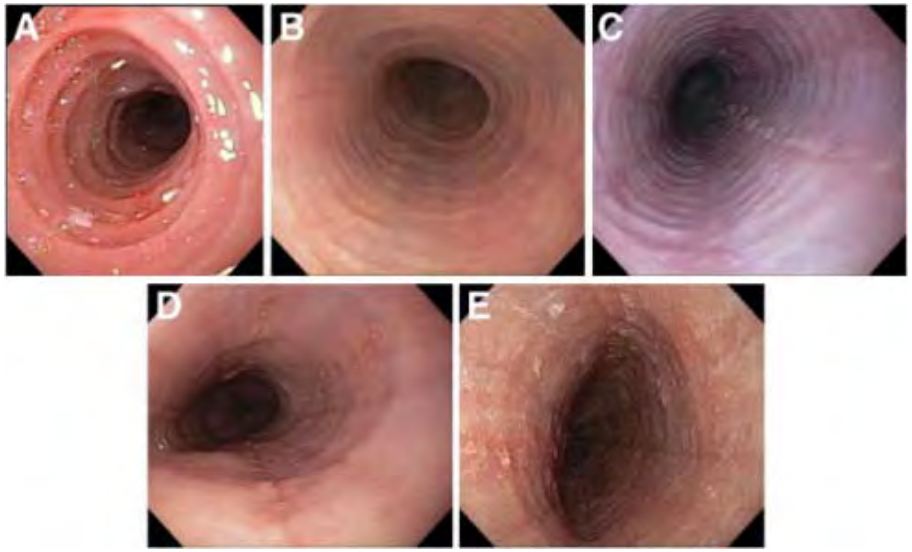
Pill Esophagitis

- Pill esophagitis often involves the esophagus at the level of the aortic arch because:
  - This is the area where the amplitude of the peristaltic wave is the lowest
  - This is the area where the density of inhibitory neurons is the highest
  - This is the area where the number of submucosal glands is normally the highest
  - This is the area where infiltration with eosinophils is the highest
- Often involves these medications
  - Antibiotics (e.g, tetracycline, doxycycline, clindamycin)
  - Ant-inflammatories (e.g., aspirin, NSAIDs esp ibuprofen – avoid HS use)
  - Bisphosphonates (e.g., alendronate)
  - Other (e.g., potassium choloride, quinidine, iron)

Pill Esophagitis

Eosinophilic Esophagitis (EoE)

- Defined by a panel of experts as "a chronic immune/antigen-mediated esophageal disease characterized clinically by symptoms related to esophageal dysfunction and histologically by eosinophil-predominant inflammation"
  - Estimated incidence 9.45/100,000
  - Estimated prevalence 55/100,000
- Should be considered in adults with a history of food impaction, persistent dysphagia, or GERD that fails to respond to medical management
- Clinical manifestations
  - Dysphagia
  - Food impaction
  - Chest pain
  - Refractory heartburn or other reflux symptoms
- Diagnosis made primarily by endoscopy and biopsy
  - Biopsies from mid/proximal and distal esophagus
  - Histology – need to differentiate from eosinophilia associated with GERD
  - Usually >15 eos per HPF
- Endoscopic features
  - Stacked circular rings
  - Strictures
  - Linear furrows
  - Small caliber esophagus



Eosinophilic Esophagitis Treatment

- Dietary therapy
  - Elimination diets
  - Allergy/immunology evaluation
- Pharmacologic
  - Topical steroids
    - Swallowed corticosteroids (fluticasone spray swallowed – adults 440-880mcg BID for 2 months)
  - Systemic steroids (if failed topical steroids)
  - PPIs (for 2 months)
- Endoscopic
  - Narrow caliber esophagus requires more careful dilatation over a guide wire
  - Perforation rate 3/1000, similar to non-EoE strictures

High Resolution Manometry In EoE

- 32% of EoE patients demonstrated pan-esophageal pressurization events with higher volume bolus challenge
- Esophageal pressurization may reflect reduced distensibility/compliance of the esophagus in EoE

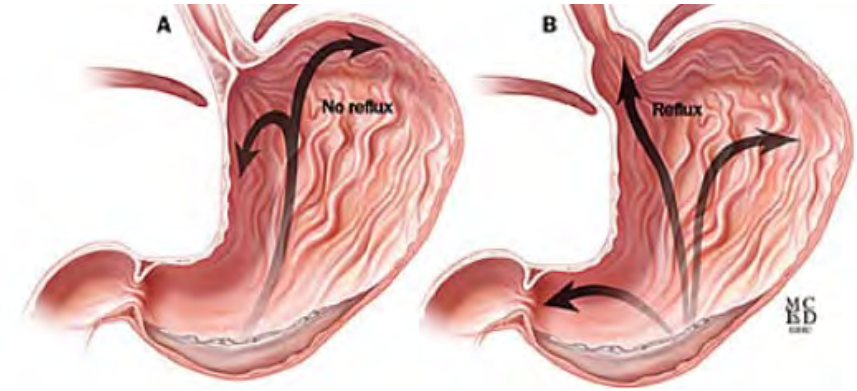
GASTROESOPHAGEAL REFLUX DISEASE (GERD)

GERD

- Anatomy/physiology
- Clinical manifestations and presentation
  - Esophageal symptoms
  - Extra-esophageal symptoms
- Diagnosis
  - Barium esophagram
  - EGD
  - pH studies (24 hour vs. BRAVO)
  - Esophageal motility
- Complications
  - Non-erosive reflux disease (NERD)
  - Acid vs. bile reflux
- Treatment
  - Medications
    - Medication side effects and complications
  - The refractory patient
    - Medical treatment (including baclofen)
    - Surgical
    - Endoscopic

GERD Introduction

- Montreal Classification defines GERD as a condition that develops when reflux of stomach contents causes troublesome symptoms or complications
- Montreal Working Group defined heartburn as troublesome if symptoms occur 2 or more days per week or moderate to severe symptoms occur more than one day a week (usually this is for >6 months)
- GERD prevalence of 10-20% in the Western world
- Over $1B annually OTC remedies, $10B PPIs



Pathophysiology Of GERD

- Primary event is movement of gastric juice from stomach into esophagus
- 1) GE junction incompetence
  - Transient LES relaxations (tLESRs) – major factor in mechanism of belching as well
    - tLESRs more frequent and for prolonged periods
    - Associated with acid reflux as opposed to gas venting
    - Can be inhibited by GABA type B agonists (baclofen)
  - Vagally mediated reflex
  - Hypotensive LES
    - Minority of patients
  - Anatomic disruption of GEJ (hiatal hernia)
  - Obesity
  - Pregnancy – 30-50% with reflux
    - Mechanical and hormonal

Pathophysiology Of GERD

- Factors which reduce LES pressure
  - Gastric distension
  - CCK
  - Foods (fat, chocolate, alcohol, caffeine)
  - Smoking
  - Drugs (e.g., nitrates, CCBs, narcotics, benzos, progesterone)
- 2) Esophageal acid clearance
  - Prolonged with esophagitis and can be prolonged with hiatal hernia
- 3) Esophageal emptying
  - Peristaltic dysfunction
  - Intra-esophageal reflux
- 4) Salivation
  - If reduced, can contribute to GERD
- 5) Esophageal sensitivity
  - Non-erosive reflux disease (NERD)

GERD Barrier

- GEJ forms anti-reflux barrier
  - Dependent on:
    - GEJ complex
    - Changes with gastric distension
    - Esophageal motility
    - Intra-abdominal pressure
    - Gravity

Diagnosis Of GERD

- Presumptive diagnosis with typical symptoms of heartburn and regurgitation
  - Can treat empirically with a PPI
  - Belching as a primary symptom often not GERD related
- Patients with non-cardiac chest pain suspected due to GERD should have cardiac cause excluded before GI evaluation
- These studies/procedures NOT required to diagnose GERD in the presence of typical GERD symptoms:
  - Barium radiographs
  - Upper endoscopy
  - Biopsies from distal esophagus
  - Esophageal manometry
  - Ambulatory reflux monitoring
  - Screening for H. pylori infection
- Endoscopy recommended for patients with alarm symptoms and for screening patients at high risk for complications
  - For patient who are acid suppressant dependent
  - Especially men over age 50
  - Repeat endoscopy not indicated for patients without Barrett's esophagus in absence of new symptoms

Extraesophageal Manifestations Of GERD

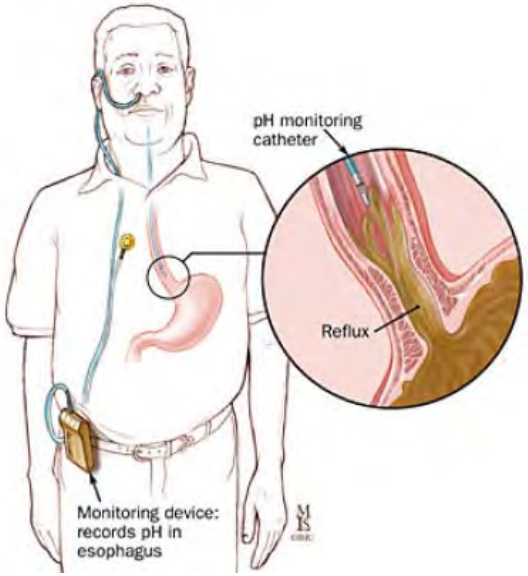
- GERD can be considered a potential co-factor in patients with:
  - Asthma
  - Chronic cough
  - Laryngitis
- PPI trial is recommended to treat extraesophageal symptoms in patients who also have typical GERD symptoms

Summary of Diagnostic Testing Evidence

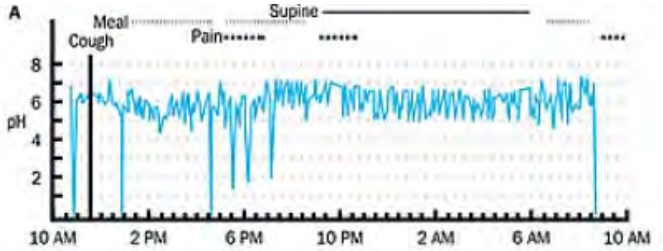
Diagnostic test	Indication	Highest level of evidence	Recommendation
PPI trial	Classic symptoms, no warning signs,	Meta-analysis	Negative trial does not rule out GERD
Barium swallow	Not for GERD diagnosis. Use for evaluation of dysphagia	Case–control	Do not use unless evaluating for complication (stricture, ring)
Endoscopy	Alarm symptoms, screening of high-risk patients, chest pain	Randomized Controlled Trial	Consider early for elderly, those at risk for Barrett's, non-cardiac chest pain, patients unresponsive to PPI
Esophageal biopsy	Exclude non-GERD causes for symptoms	Case–Control	Not indicated for diagnosis of GERD
Esophageal manometry	Preoperative evaluation for surgery	Observational	Not recommended for GERD diagnosis. Rule out achalasia/ scleroderma-like esophagus preop
Ambulatory reflux monitoring	Preoperatively for non-erosive disease. refractory GERD symptoms, GERD diagnosis in question	Observational	Correlate symptoms with reflux, document abnormal acid exposure or reflux frequency

Continuous pH Monitoring

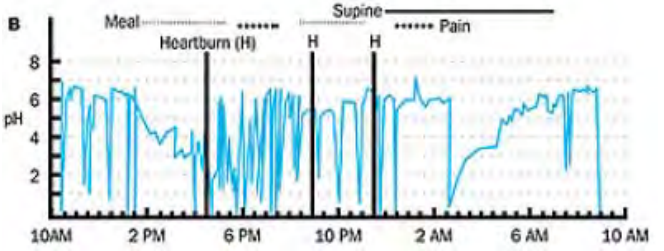
- Acid-sensitive catheter is placed in the esophagus and is attached to a small monitoring device
- Changes in esophageal pH are recorded over an extended period of time (up to 24 hours)
- Provides information on the severity and pattern of reflux
- Considered the best test for the diagnosis of GERD, however there is a 10-20% false negative rate
- If intra-esophageal pH is < 4 for more than 10% of the time, patient is considered to have pathologic reflux



Physiologic Reflux



Pathologic Reflux



Multichannel Intraluminal Impedance (MII) Testing

- MII is a catheter-based method to detect intraluminal bolus movement within the esophagus
- The principal of impedance testing is based on a measurement of changes in resistance to electrical current when a bolus passes by a pair of metallic rings mounted on a catheter
- Liquid-containing boluses with an increased number of ions and higher conductivity will lower the impedance to a nadir value
- Impedance returns to baseline once bolus is cleared by a contraction
- Can be performed in combination with manometry or pH testing
- When combined with manometry, bolus transit data can add a functional analysis to manometrically recorded contractions
- When combined with pH testing, it allows for the detection of GE reflux independent of pH (i.e., both acid and non-acid reflux)

Indications For MII-EM

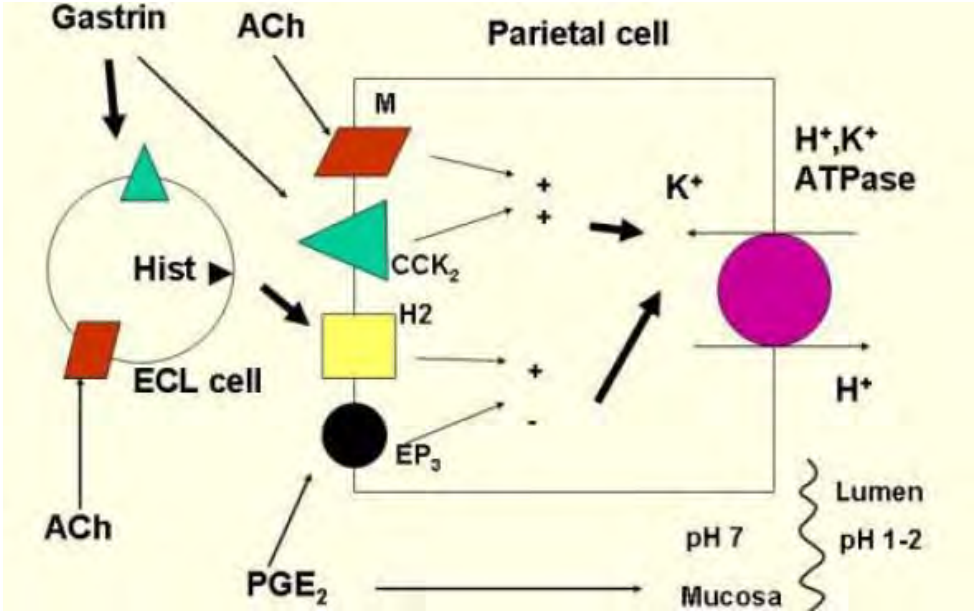
- Similar to those for esophageal manometry
  - Evaluation of patients with dysphagia, non-cardiac chest pain, or heartburn regurgitation
  - Pre-op evaluation prior to anti-reflux procedures (surgical or endoscopic)
  - Location of the LES prior to pH catheter placement
- MII-pH can be performed on or off PPI therapy, but for diagnostic purposes in the refractory patient, stopping acid suppression therapy for 1-2 weeks may add additional diagnostic value

Physiology Of Gastric Acid Secretion

- Normal stomach pH
  - Basal: pH 1-2
  - Post prandial (1 hour): pH 4-5

- Parietal cell
  - Primary acid producing cell
  - Located in body and fundus of stomach
  - H+/K+ ATPase pump
    - Generates largest ion gradient known in vertebrates
  - Influenced by acetylcholine, histamine, gastrin, prostaglandins

Gastric Acid Secretion



Pharmacologic Management Of GERD

- **Antacids**: local neutralization of acid
  - Aluminum hydroxides
  - Magnesium hydroxides
  - Calcium carbonate
  - Sodium bicarbonate
  - Gaviscon (aluminum hydroxide + magnesium carbonate)
- **H2 receptor antagonists** (H2RAs): compete with histamine for binding to H2 receptors on parietal cells
  - Cimetidine
  - Ranitidine
  - Famotidine
  - Nizatidine
- **Proton pump inhibitors** (PPIs): prodrugs that require activation by an acidic environment (symptom improvement occurs with pH<4)
  - Half-life 1-2 hours
  - Full effect may take up to 2-5 days
  - Duration of effect 24-48 hours, but can affect acid secretion for up to 7-14 days
  - Some patients with defective proton pumps may respond better to H2RAs
  - Omeprazole
  - Lansoprazole
  - Rabeprazole
  - Pantoprazole
  - Esomeprazole
- **Cytoprotectants**
  - Sucralfate
  - Misoprostol
  - Bismuth compounds

Management Of GERD

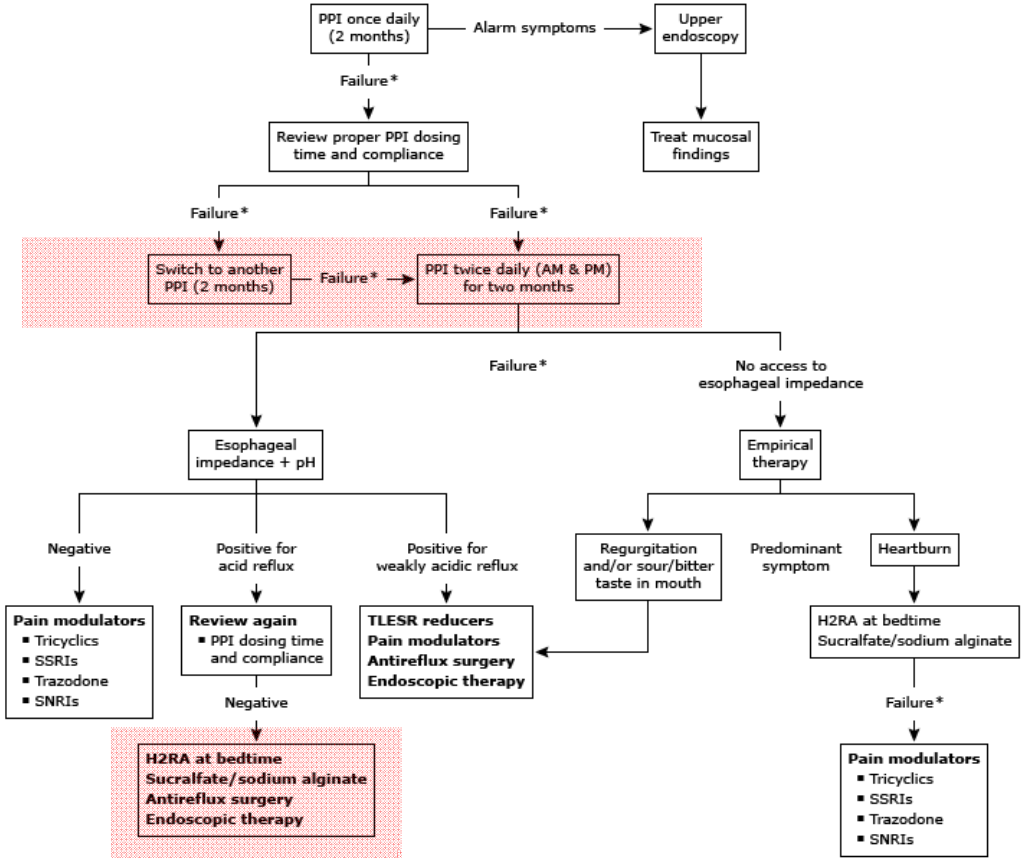
- Lifestyle
  - Weight loss for overweight patients or those with recent weight gain
  - Head of bed elevation and avoidance of meals 2-3 hours before bedtime
  - Routine global elimination of food that can trigger reflux is not recommended
- Medication
  - An 8-week course of PPIs is the therapy of choice for symptom relief and healing of erosive esophagitis
  - PPI therapy should be initiated at once a day dosing before the first meal of the day
  - Twice daily dosing, a different PPI, and/or adjustment of dose timing should be considered for patients with a partial response to initial PPI therapy
  - Maintenance PPI therapy should be administered for patients who continue to have symptoms after PPI is discontinued and in patients with complications including erosive esophagitis and Barrett's esophagus
    - For long-term PPI therapy, lowest effective dose and on-demand/intermittent therapy should be used if possible
  - H2-receptor antagonist (H2RA) therapy can be used as a maintenance option in patients without erosive disease if patients experience heartburn relief
  - Bedtime H2RA therapy can be added to daytime PPI therapy in selected patients with night-time reflux
  - Therapy for GERD other than acid suppression should not be used in GERD patients without diagnostic evaluation
  - H2RAs safe in pregnant patients if clinically indicated; PPIs only if benefit outweighs risk

Potential Risks Associated With PPIs

- Osteoporosis – especially in post-menopausal women; need for calcium and vitamin D supplementation in patients on long-term therapy
- PPI therapy can be a risk factor for Clostridium difficile infection and should be used with care in patients at risk
- Short-term PPI use may increase the risk of community-acquired pneumonia
  - Risk does not appear elevated in long-term users
- Potential for malabsorption
  - Magnesium
  - Calcium
  - Vitamin B12
  - Iron
- Atrophic gastritis in over 30% of patients with chronic use
  - Theoretical increased risk of gastric cancer
- Gastric polyps
- Associated with kidney disease
  - Acute interstitial nephritis
  - Chronic kidney disease
- Associated with dementia
- PPI therapy does not need to be altered in concomitant clopidogrel users

Management of GERD Refractory To PPI Therapy

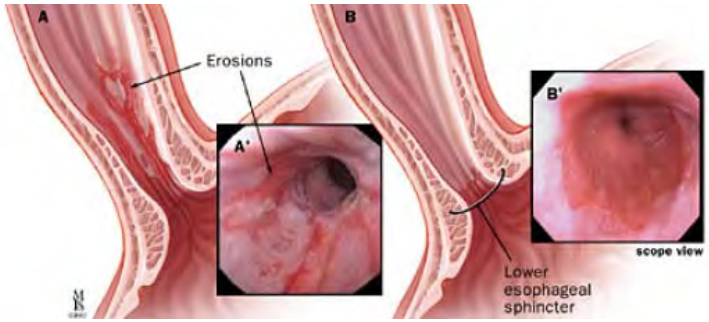
- Optimize PPI therapy
- Perform upper endoscopy to exclude non-GERD etiologies
- For patients with persistent extraesophageal symptoms, assessment for other etiologies through concomitant evaluation by ENT, pulmonary, or allergy specialists
- For patients with negative evaluation, would perform ambulatory reflux monitoring
  - Reflux monitoring off medication can be performed by any available modality
  - Testing on medication should be performed with impedance-pH monitoring to enable measurement of nonacid reflux
- Refractory patients with objective evidence of ongoing reflux as cause of symptoms should be considered for additional antireflux therapies that may include surgery or transient lower esophageal sphincter relaxation (TLESR) inhibitors (e.g., baclofen)



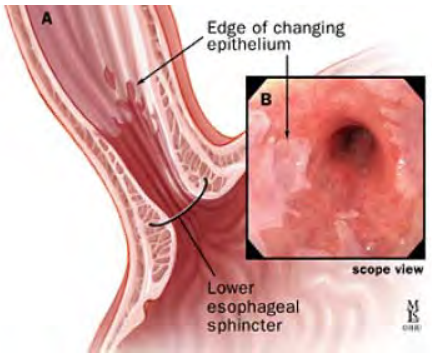
GERD Complications

- Esophagitis
  - Presence of inflammatory cells within the esophageal mucosa
  - Ranges from microscopic changes in biopsies (microscopic esophagitis) to inflamed mucosa without erosion (nonerosive esophagitis) to frankly eroded or ulcerated mucosa (erosive esophagitis)
  - Severity of esophagitis not well correlated with severity of GERD symptoms
  - LA Classification (Grade A-D)
- Barrett's esophagus
  - Length of distal esophagus is covered by an abnormal-looking cellular lining
  - Consequence of abnormal healing of erosive esophagitis
  - Associated with an increased risk of developing esophageal cancer
- Esophageal stricture
  - Narrowing of the esophagus due to the healing process of ulcerative esophagitis
  - May result in solid food dysphagia and episodic food impaction

Erosive Esophagitis



Barrett's Esophagus

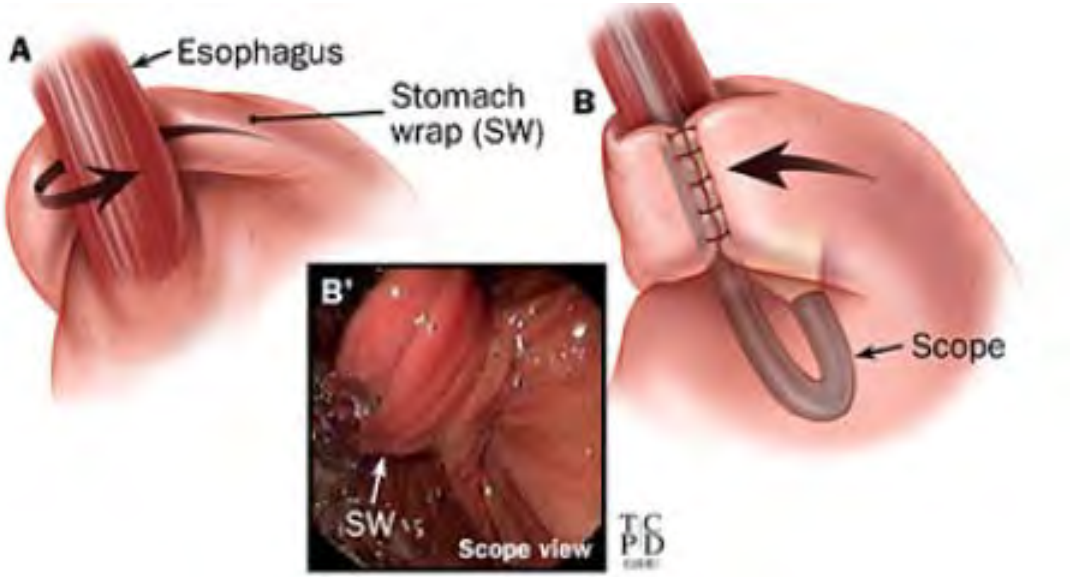


Esophageal Stricture

Surgical Therapy Of GERD

- A number of surgical approaches have been advocated
- All involve an attempt to bolster the strength of the antireflux barrier
- The most commonly employed surgical approach is referred to as a Nissen fundoplication

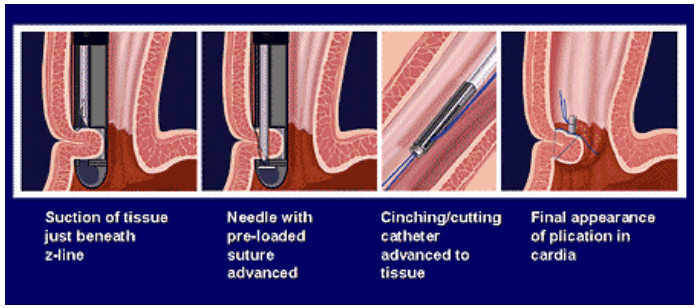
Nissen Fundoplication



Endoscopic And Minimally Invasive GERD Therapy

- Still considered experimental
  - Suturing devices (e.g., EndoCinch)
  - Radiofrequency ablation (e.g., Stretta)
  - Implants/injections (e.g., LINX)
  - Neurostimulation (e.g., EndoStim)

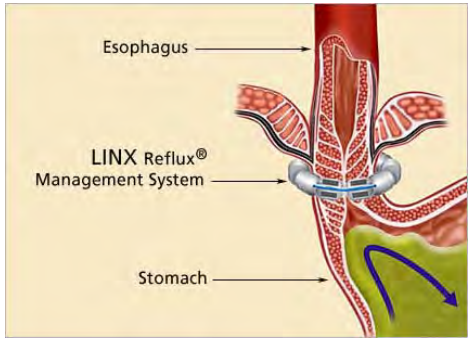
EndoCinch



EndoStim

Stretta

LINX



Extraesophageal Manifestations of GERD - Cough

- Cough
  - GERD is often reported to be the 2nd or 3rd most common cause of persistent cough (and most common in some reports)
  - Heartburn or sour taste in mouth absent in more than 40% of patients in whom cough is due to reflux
  - Several factors potentially responsible for cough due to GERD
    - Stimulation of receptors in the upper respiratory tract
    - Aspiration of gastric contents (acid, pepsin), leading to stimulation of receptors in the lower respiratory tract
    - Esophageal-tracheobronchial cough reflex induced by acid reflux into the distal esophagus
  - GERD can also contribute to asthma symptoms

Laryngopharyngeal Reflux (LPR)

- Retrograde movement of gastric contents into the laryngopharynx leading to symptoms referable to larynx/hypopharynx
- Symptoms include dysphonia/hoarseness, globus, mild dysphagia, chronic cough, nonproductive throat clearing
- Primarily an UES problem that mainly occurs in upright position during periods of physical exertion (e.g., bending over, Valsava, exercise)
- Distinct clinical entity from GERD (GERD mainly a problem of the LES)
- Much less acid exposure is necessary to create LPR compared to GERD
- Most patients relatively unaware of LPR, with only 35% reporting heartburn

LPR Treatment

- Drug therapy
  - Acid suppression
    - PPIs
    - H2 Blockers
    - Antacids
  - Neuromodulating agents
    - Tricyclic antidepressants
      - Nortriptylane
    - Gabapentin
    - Pregabalin

Extraesophageal Manifestations of GERD - Globus

- Globus sensation
  - Functional esophageal disorder characterized by a sensation of a lump or foreign body in the throat
  - Also referred to as globus pharyngeus or globus hystericus
  - Unclear pathogenesis, but etiologies include:
    - Visceral hypersensitivity
    - Abnormalities of the UES
    - Psychologic and psychiatric disorders
    - GERD

Treatment Of Globus

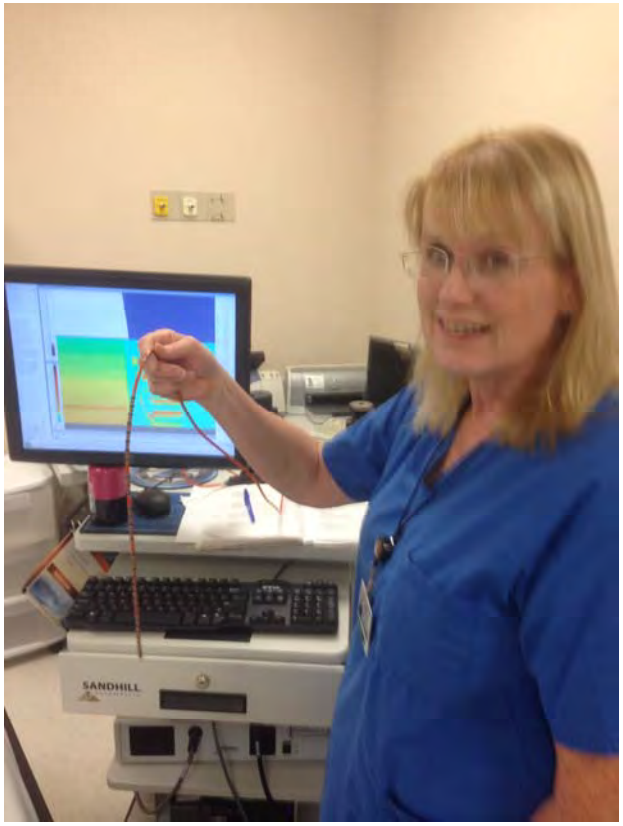
- Conservative therapy
  - Reassurance that globus is a benign disorder
- Acid suppression
  - 6-8 weeks of PPI therapy
  - 1/3 of patients experience partial relief
- Antidepressants (e.g., amitriptyline)
- Other
  - Gabapentin
  - Relaxation therapy

Additional Evaluation Of Globus

- Additional evaluation warranted in patients with recurrent or persistent symptoms despite conservative management or those with alarm features (e.g., pain, lateralization of symptoms, dysphagia, odynophagia, weight loss, change in voice, neck mass, unexplained cervical adenopathy)
- Modalities include:
  - Nasoendoscopy
  - Videofluoroscopy
  - Barium swallow with solid bolus (e.g., barium tablet)
  - Esophageal manometry
  - Esophageal pH and impedance
  - Upper endoscopy

MOTILITY DISORDERS OF THE ESOPHAGUS

Introduction



Motility Disorders

- Presentation
  - Dysphagia
  - Reflux
  - Cough
  - Choking
- Oropharyngeal dysphagia
- Causes
  - Neurologic and neuromuscular disorders
  - Cricopharyngeal dysfunction
  - Disorders that affect the esophageal body

Major Disorders Of Esophageal Peristalsis

- Achalasia
- Hypertensive LES/EGJ outflow obstruction
- Hypertensive peristaltic disorders
  - Nutcracker esophagus
  - Jackhammer esophagus ("spastic nutcracker")
- Distal esophageal spasm (DES)
- Ineffective esophageal motility (IEM)
- Non-specific motor disorders (e.g., secondary to diabetes)

Achalasia

- Most common esophageal motor disorder
  - Incidence 1.6 cases/100,000
  - Prevalence 10 cases/100,000
- Results from inflammation and degeneration of neurons (myenteric plexus in esophageal wall) – possible viral etiology
- Loss of inhibitory neurons in esophagus results in increased LESP (not required) and more importantly inability of LES to relax to baseline
- Aperistalsis
- Dysphagia mainly result of defect in LES relaxation

Hypertensive LES

- Presentation
  - Chest pain/dysphagia/globus
  - May be an achalasia variant
- Diagnosis
  - LES pressure >35mmHg and failure to relax below IRP of 15mmHg
  - Normal peristalsis
  - More important than pressures: failure of full relaxation of LES (incomplete bolus transfer)
  - Can overlap with other spastic esophageal conditions
    - May need additional provocation (bread swallows, multiple rapid swallows, solid swallows)
    - EUS recommended prior to therapy to exclude infiltrative or compressive disease (e.g., malignancy)
- Treatment
  - Balloon dilation or Botox injection
  - POEM

Jackhammer Esophagus

- Also known as hypercontractile esophagus or spastic nutcracker esophagus
- Offshoot of nutcracker esophagus
  - 4% of manometry referrals; rule out mechanical obstruction
- Presentation
  - Chest pain/dysphagia
- Diagnosis
  - At least one DCI > 8000
  - Repeated high amplitude contractions
  - Normal DL (≥ 4.5 sec)
- Treatment
  - Trial of nitrates (SL or oral) + PPI
  - Calcium channel blockers (diltiazem) PRN
  - Sildenafil PRN
  - Tricyclic antidepressants

Distal Esophageal Spasm (DES)

- Unknown etiology; likely related to defects in inhibitory neural pathways of esophagus
  - Rarest manometric diagnosis (3%)
  - Classic corkscrew esophagus very rare
- Presentation
  - Chest pain/dysphagia
  - Symptom correlation poor
- Diagnosis
  - Normal median IRP (LES relaxation), ≥ 20% premature contractions with DCI > 450mmHg x s x cm
  - Some normal peristalsis may be present
- Treatment
  - PPI + Botox injection
  - Surgical myotomy (when all else fails)

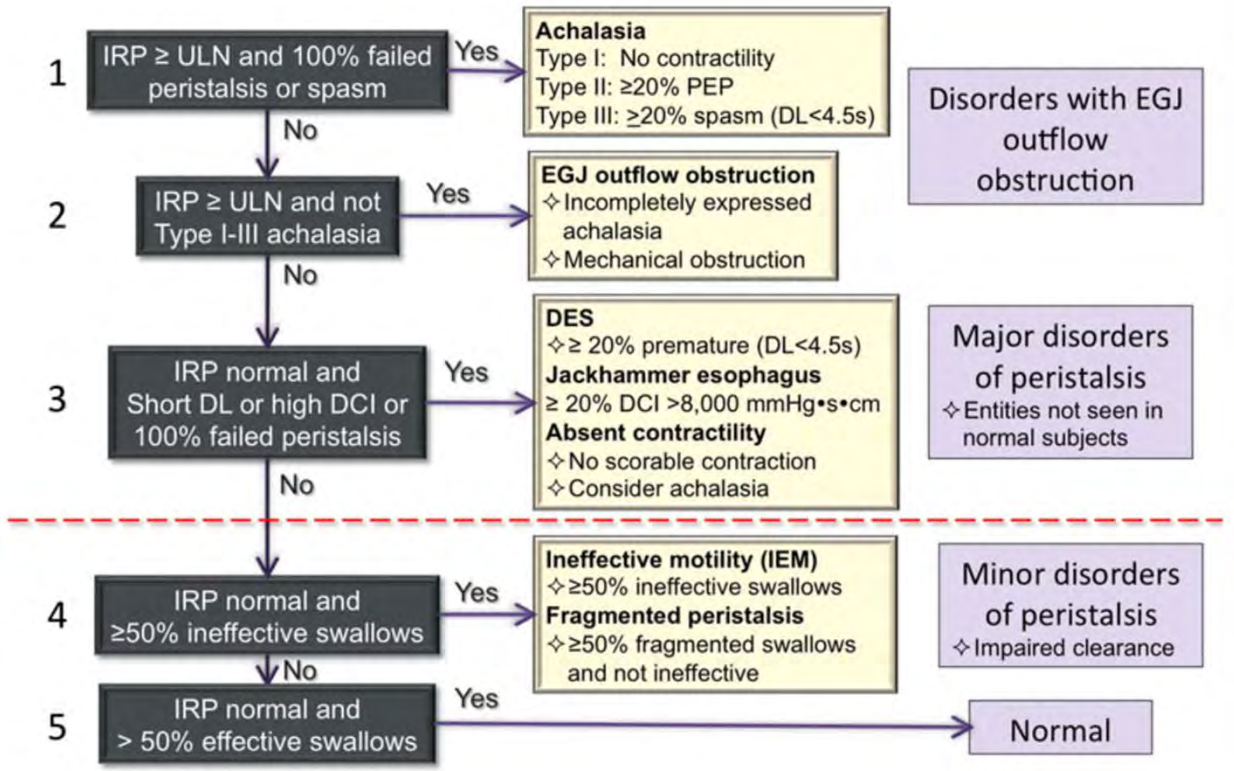
Esophageal Manometry - Indications

- Esophageal dysphagia
- Non-cardiac chest pain
- Prior to anti-reflux surgery and consider before esophageal surgery where dysphagia could result if impaired esophageal function (e.g., hiatal hernia repair)
- For certain diseases of smooth muscle or autonomic nervous system
  - Scleroderma
  - Intestinal pseudo-obstruction

The Chicago Classification

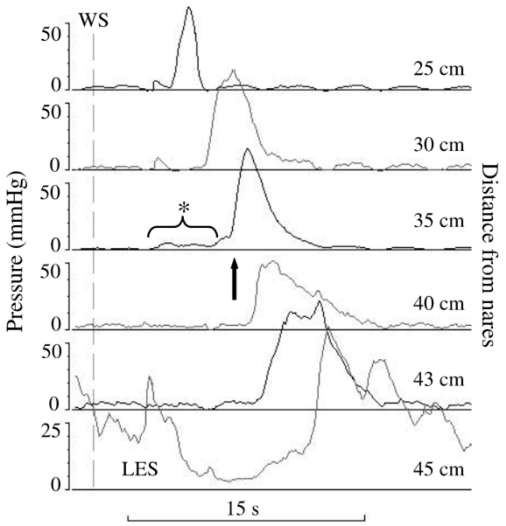
- Practical classification for esophageal motility disorders based on HRM
- Focus on disorders of the LES relaxation as a fundamental problem
- Concept to prioritize identified dysfunction into 3 subgroups
  - Achalasia/EGJ dysfunction
  - Motility patterns never seen in normal people
  - Peristaltic abnormalities out of range of normal values (<5th or >95th percentile)

The Chicago Classification

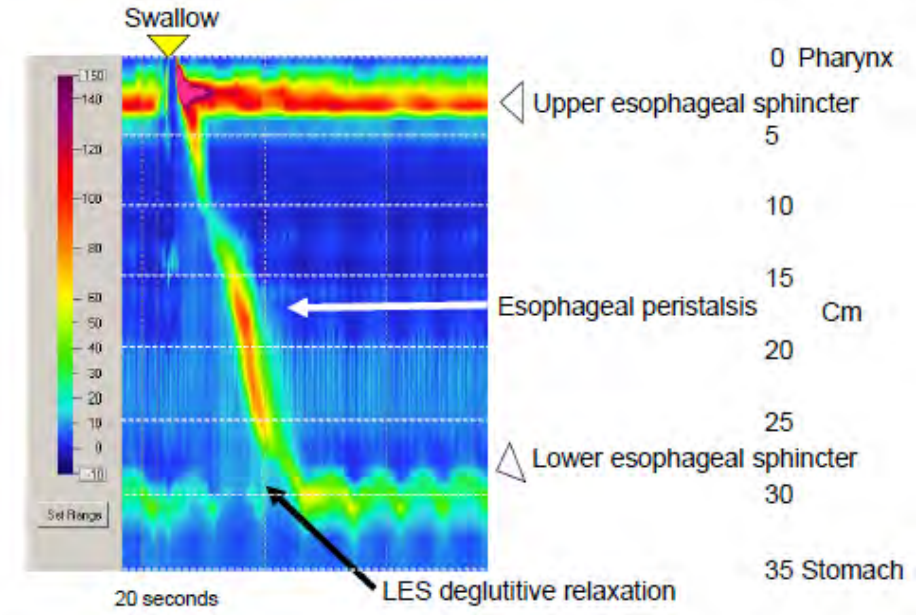


Esophageal Manometry

Normal Function

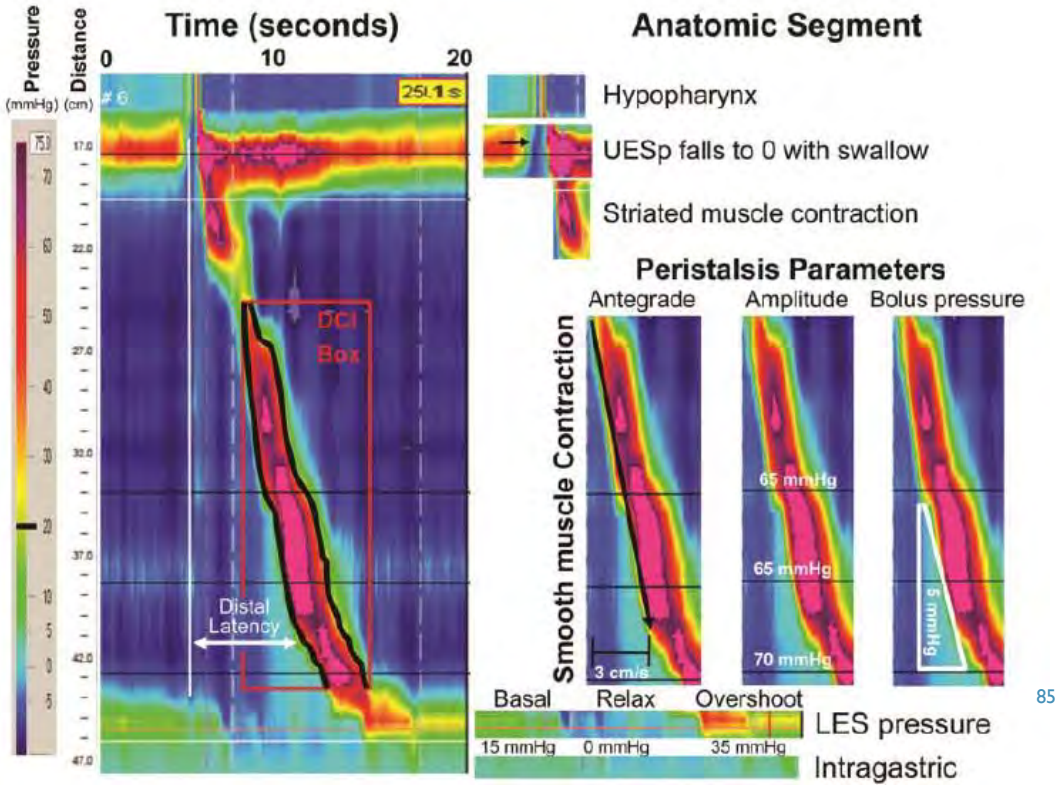


High Resolution Manometry

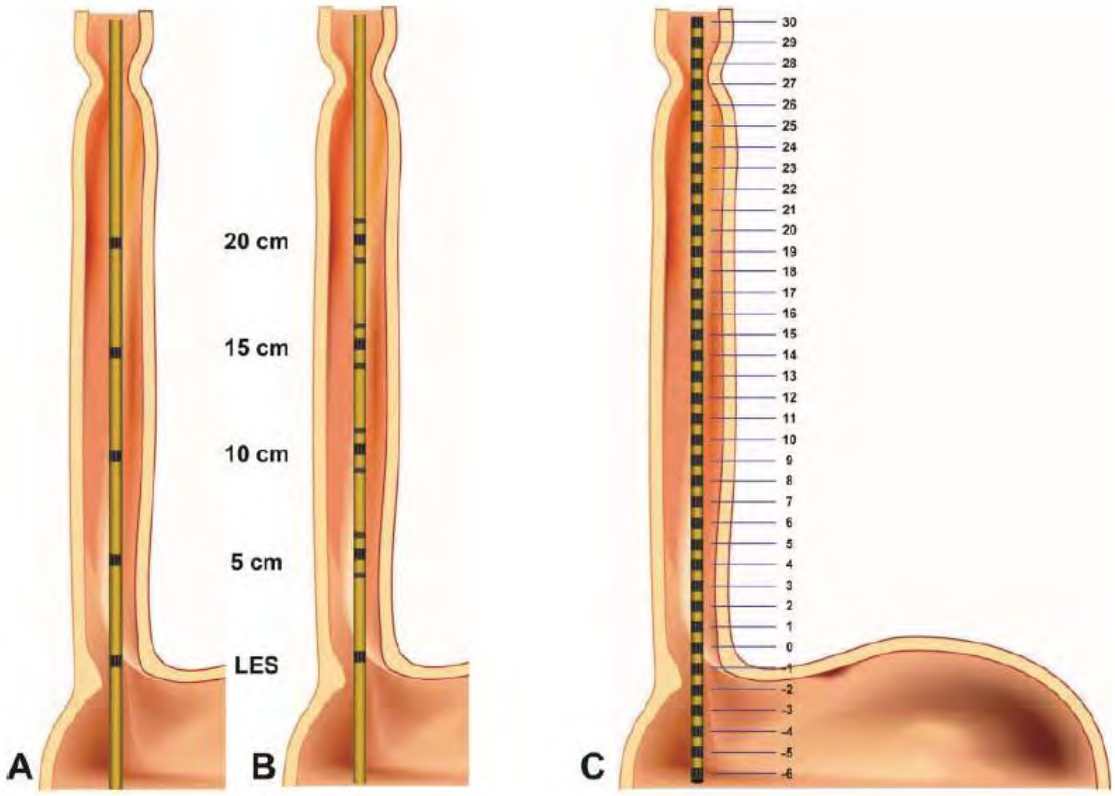


High Resolution Manometry

- Metrics
  - Integrated relaxation pressure (IRP)
    - Assesses adequacy of EGJ relaxation
  - Distal latency (DL)
    - Measure of peristaltic timing
    - Defines interval between UES relaxation and contractile deceleration point (CDP)
  - Distal contractile integral (DCI)
    - Summary measure of the vigor of distal esophageal contractions – contractile amplitude, length, and duration

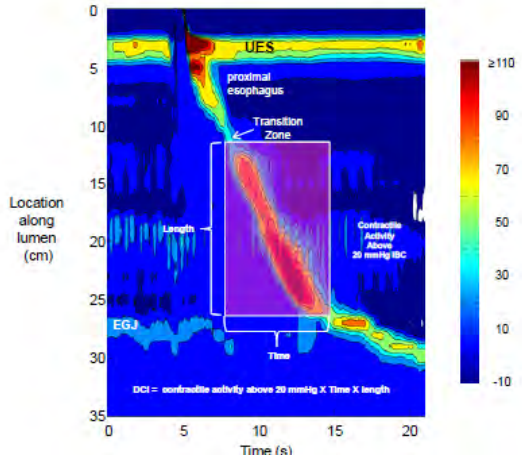


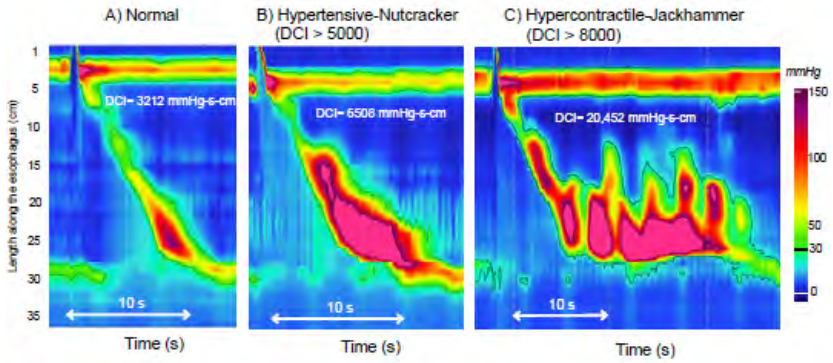
Manometry Catheter Evolution (1960-2010)



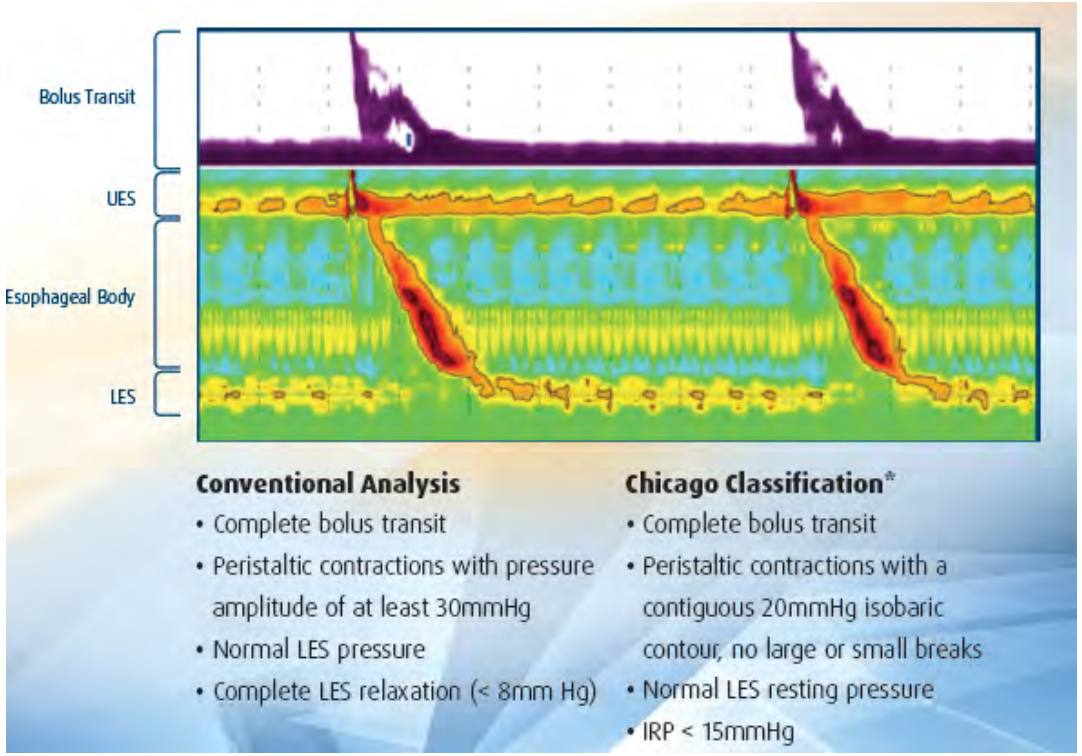
Pressure Topography Of Esophageal Motility

- Pressure magnitude converted into a color scale
  - Cold colors indicate low pressures
  - Hot colors indicate higher pressures
- Defines important anatomical landmarks and abnormalities
- Refines measurement of important motor events
  - EGJ relaxation
  - Peristaltic timing velocity
  - Contractile activity/force/amplitude
- Defines intra-luminal pressurization patterns
- Permits pattern recognition

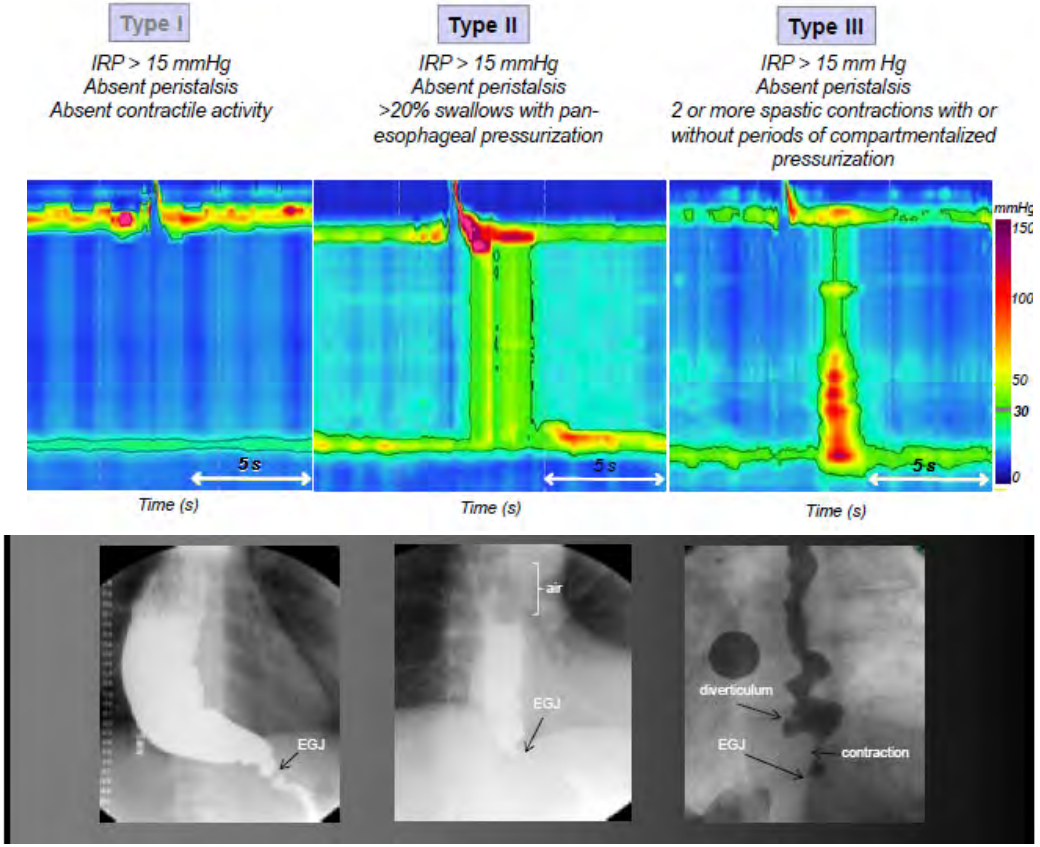




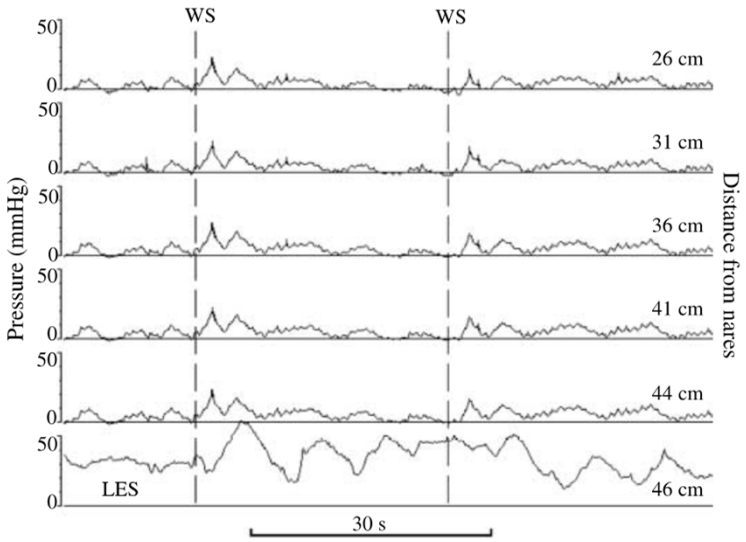
Normal Esophageal Function

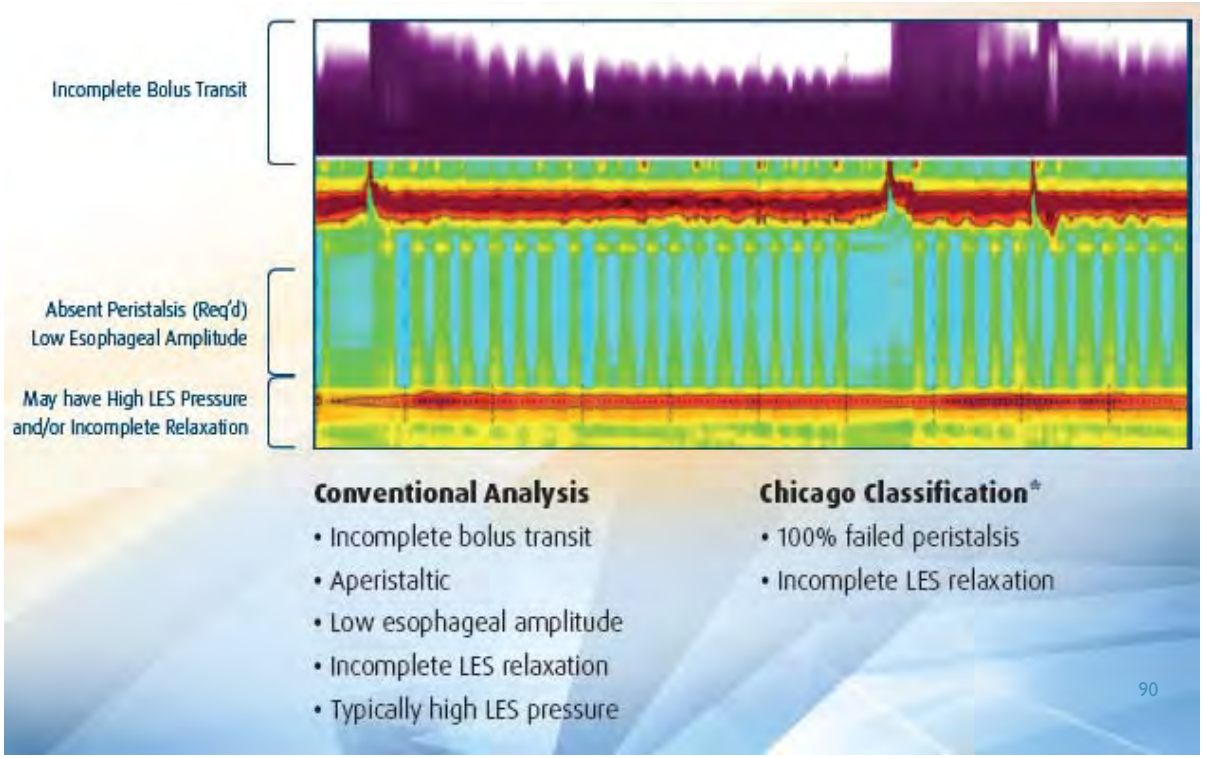


Achalasia Subtypes

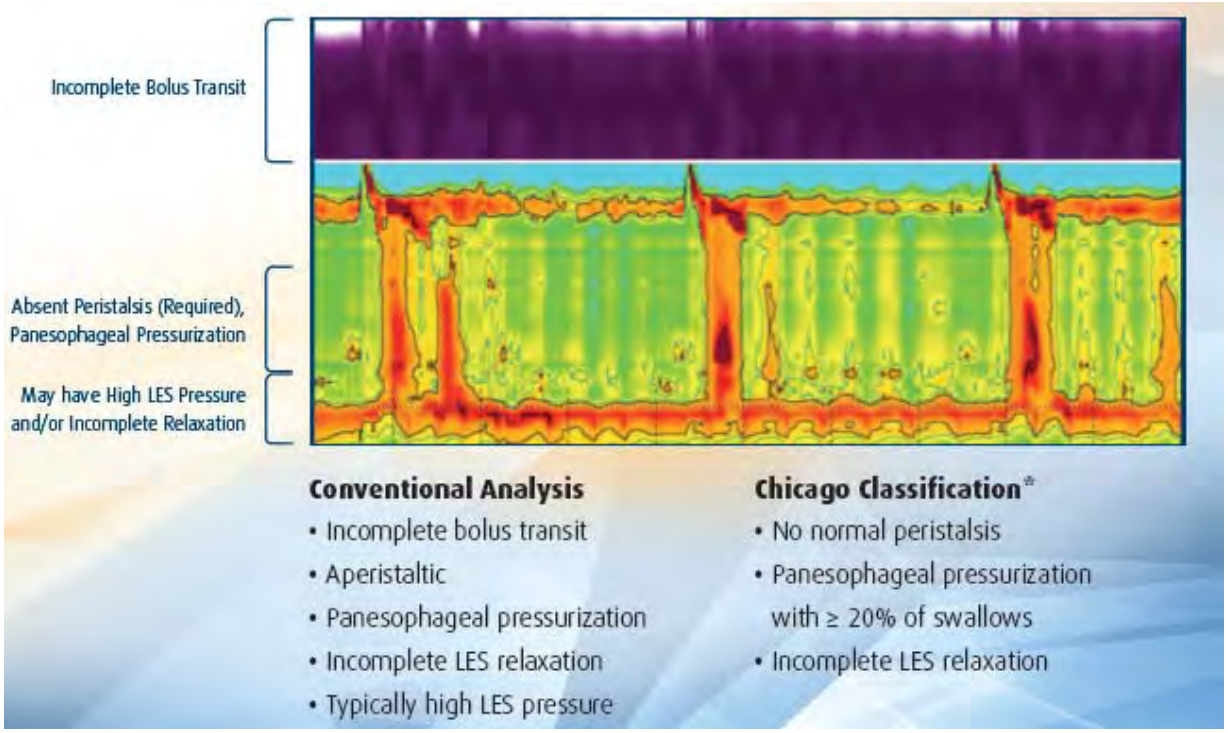


Achalasia Type I

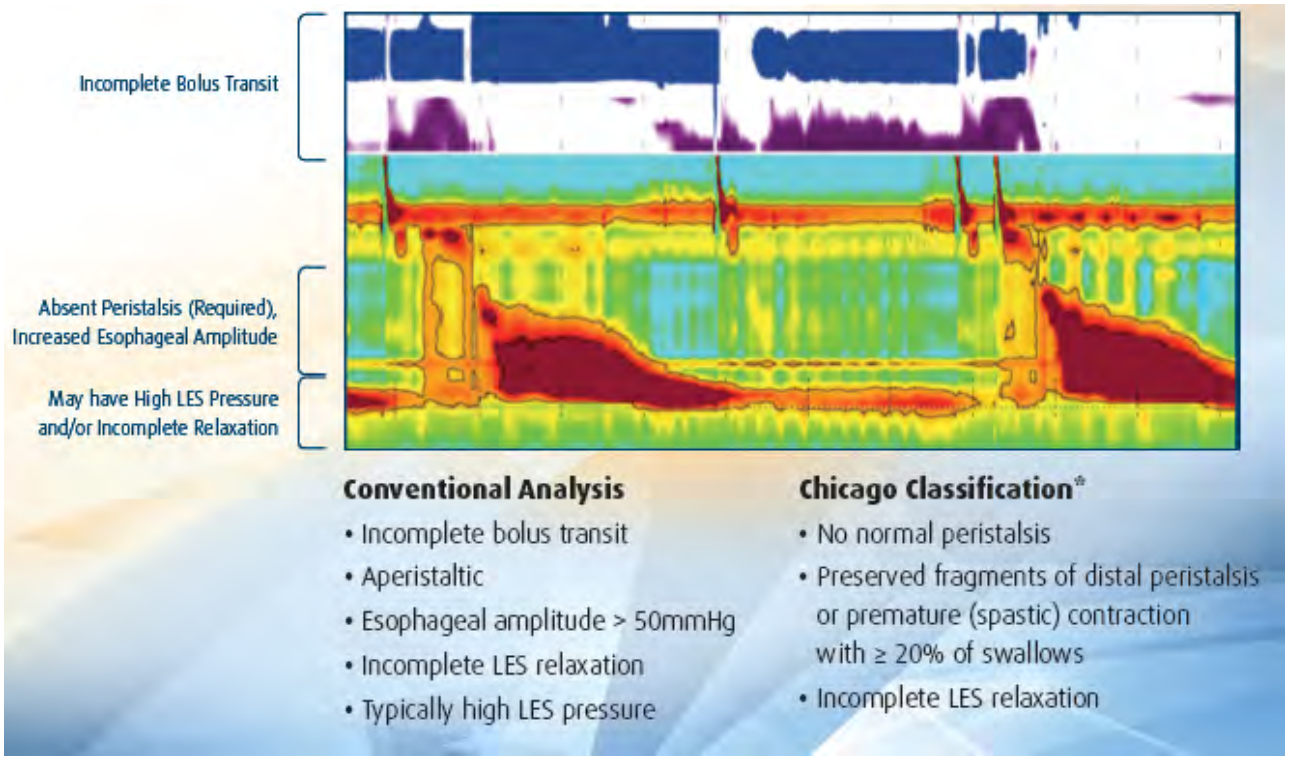




Achalasia Type II



Achalasia Type III



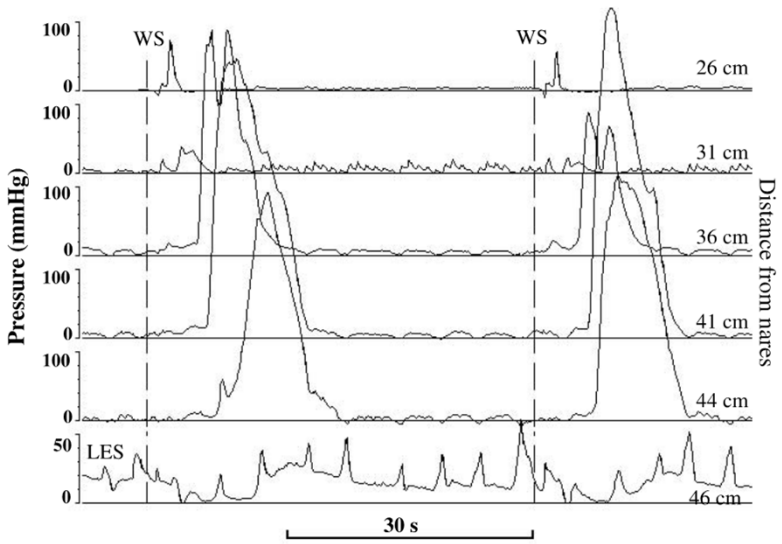
Conventional Analysis

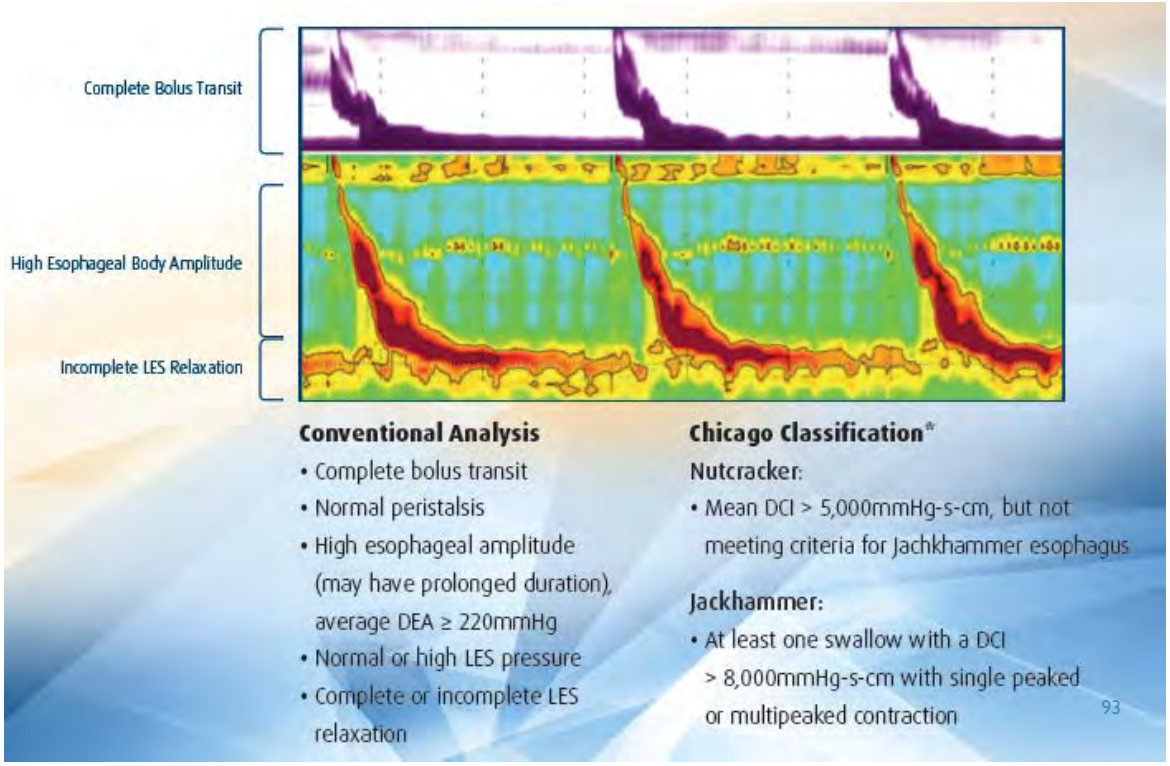
- Incomplete bolus transit
- Aperistaltic
- Esophageal amplitude > 50mmHg
- Incomplete LES relaxation
- Typically high LES pressure

Chicago Classification*

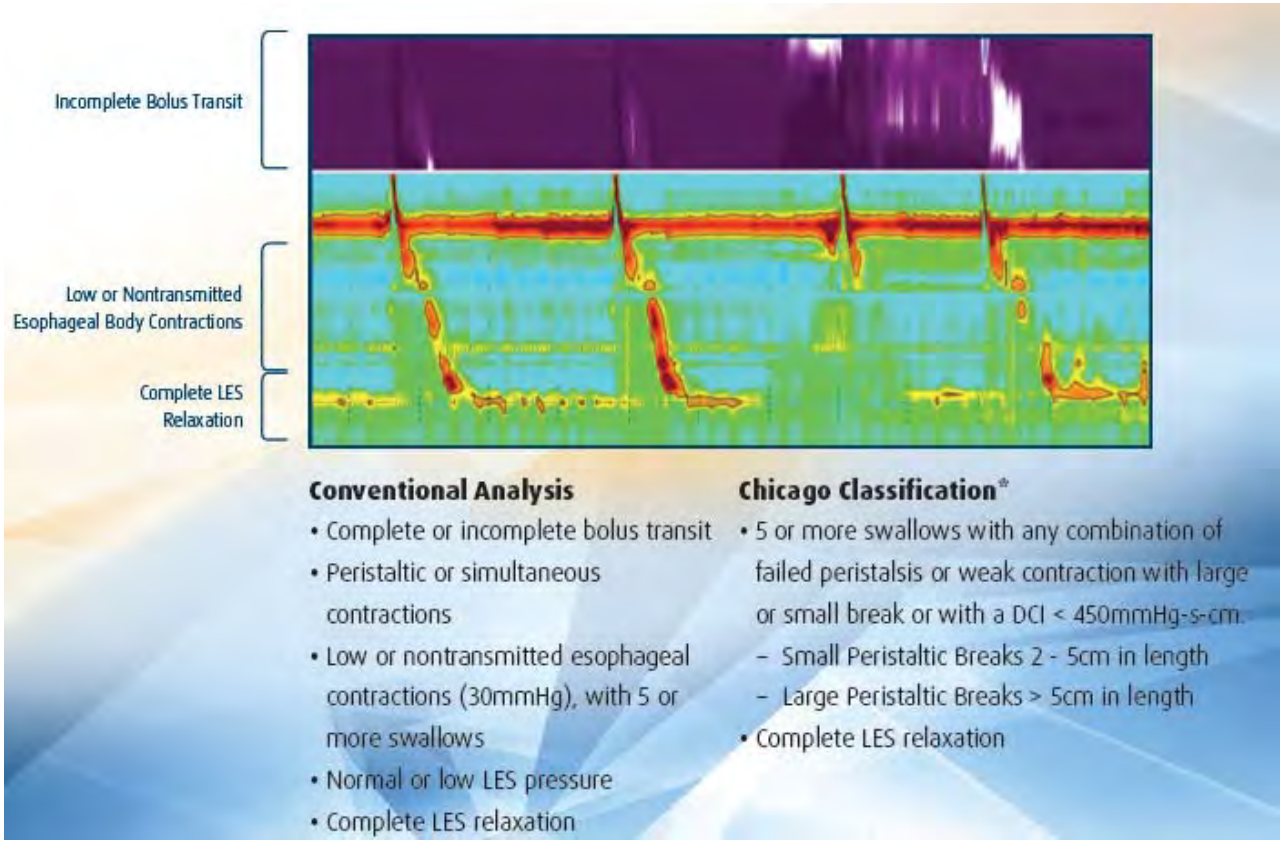
- No normal peristalsis
- Preserved fragments of distal peristalsis or premature (spastic) contraction with ≥ 20% of swallows
- Incomplete LES relaxation

Hypertensive Disorders: Nutcracker, Jackhammer





Weak/Ineffective Esophageal Motility (IEM)



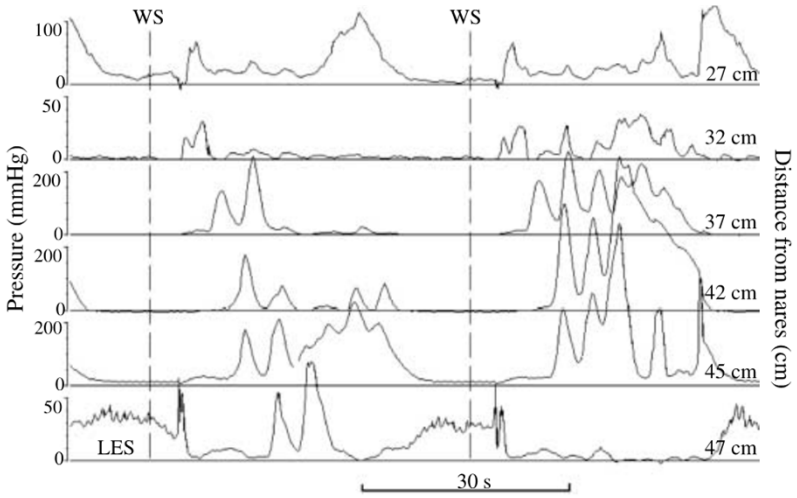
Conventional Analysis

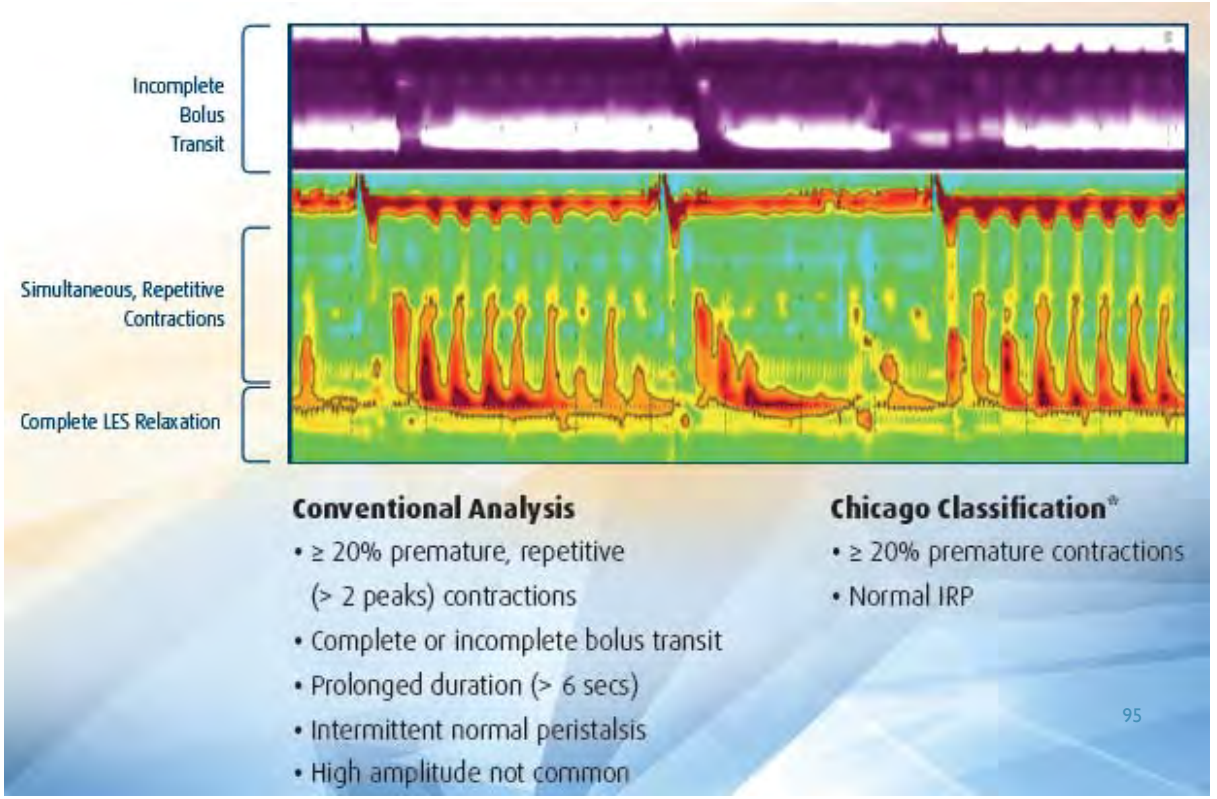
- Complete or incomplete bolus transit
- Peristaltic or simultaneous contractions
- Low or nontransmitted esophageal contractions (30mmHg), with 5 or more swallows
- Normal or low LES pressure
- Complete LES relaxation

Chicago Classification*

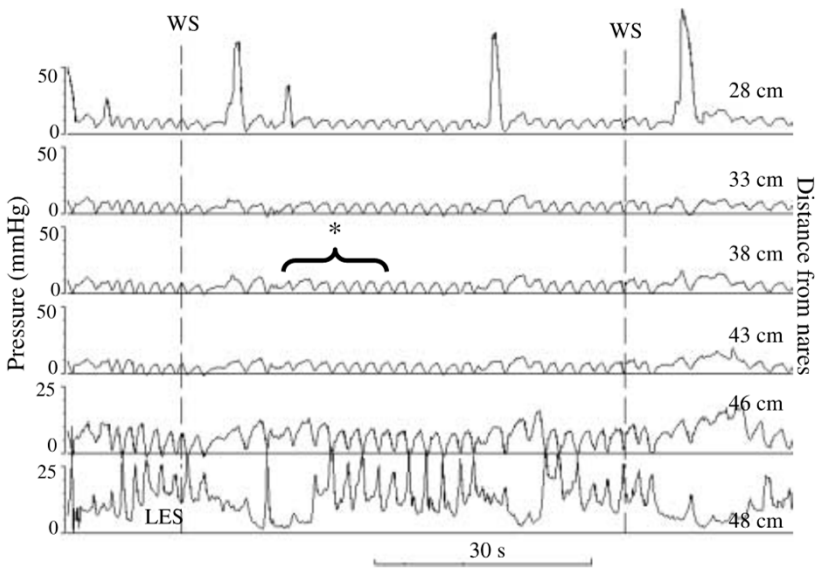
- 5 or more swallows with any combination of failed peristalsis or weak contraction with large or small break or with a DCI < 450mmHg-s-cm
  - Small Peristaltic Breaks 2 - 5cm in length
  - Large Peristaltic Breaks > 5cm in length
- Complete LES relaxation

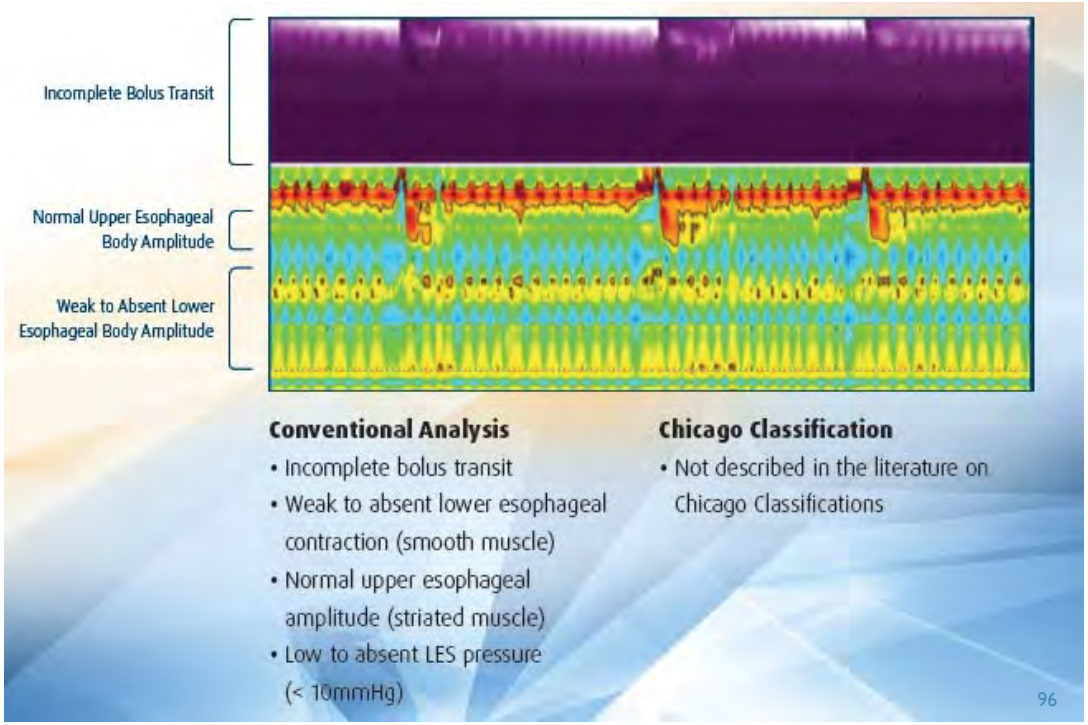
Distal Esophageal Spasm (DES)





Scleroderma



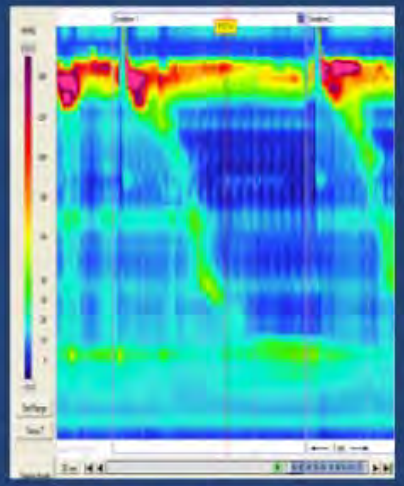


Impedance Manometry

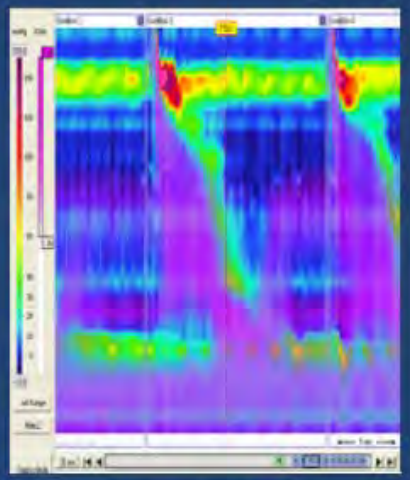
Impedance Manometry

- Addition of impedance feature allows information regarding bolus transit to be obtained
- Can identify reflux after a swallow
- Good correlation with timed barium esophagram
  - Tested in 20 achalasia patients
  - 200 ml of barium (barium esophagram) vs 200 ml of saline (impedance manometry) timed at 1 & 5 minutes

Cho Y:2014:Am Jo of Gastro



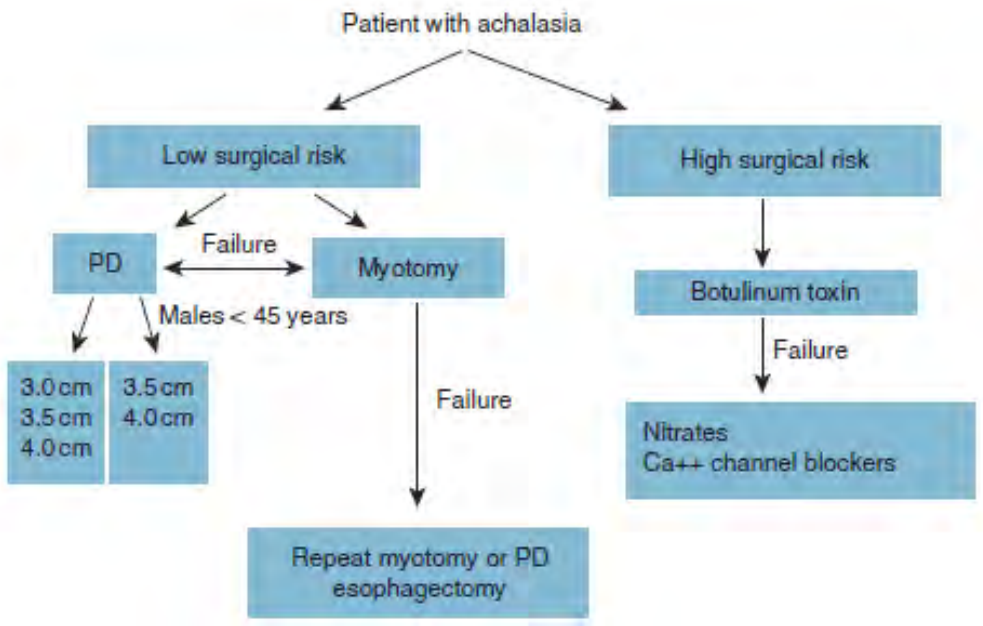
HRM without impedance



HRM with impedance

Management Of Achalasia

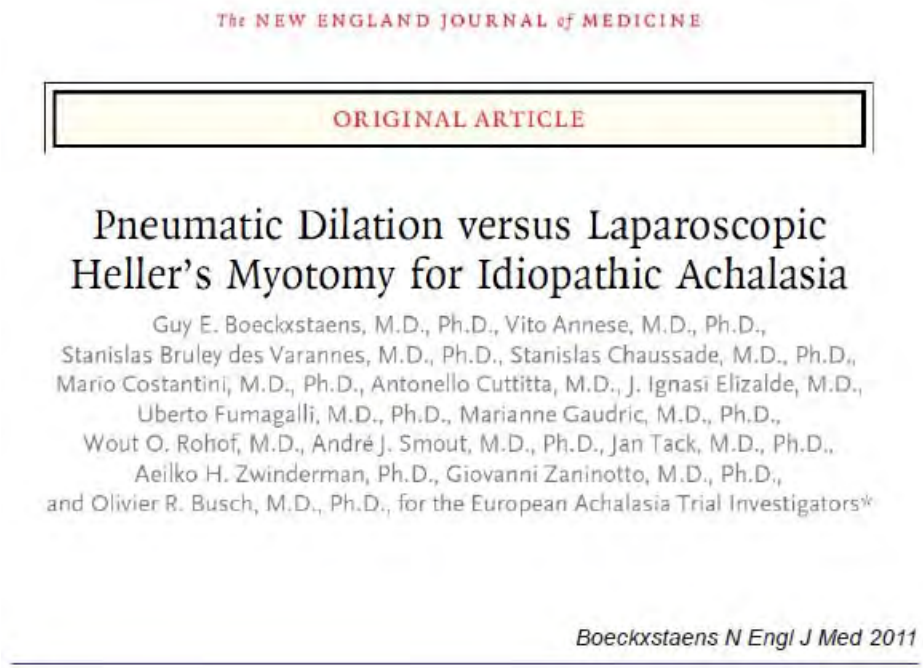
- Smooth muscle relaxants
- Botulinum toxin
- Pneumatic dilation
- Per oral endoscopic myotomy (POEM)
- Esophageal stent
- Percutaneous gastrostomy tube
- Heller myotomy



PD = pneumatic dilation

Management Of Achalasia – Pneumatic Dilation vs. Heller's Myotomy

- Pneumatic dilation protocol
  - Rigiflex balloon
  - 3cm at 5 psi x 1 min followed by 8 psi x 1 min
  - 1-3 weeks later 3.5cm
  - 4 weeks later, 4cm if Eckardt score >3
  - Repeat 3.5-4cm if recurrence during follow-up
- Heller's myotomy
  - Laparoscopic approach with Dor fundoplication



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Pneumatic Dilation versus Laparoscopic Heller's Myotomy for Idiopathic Achalasia

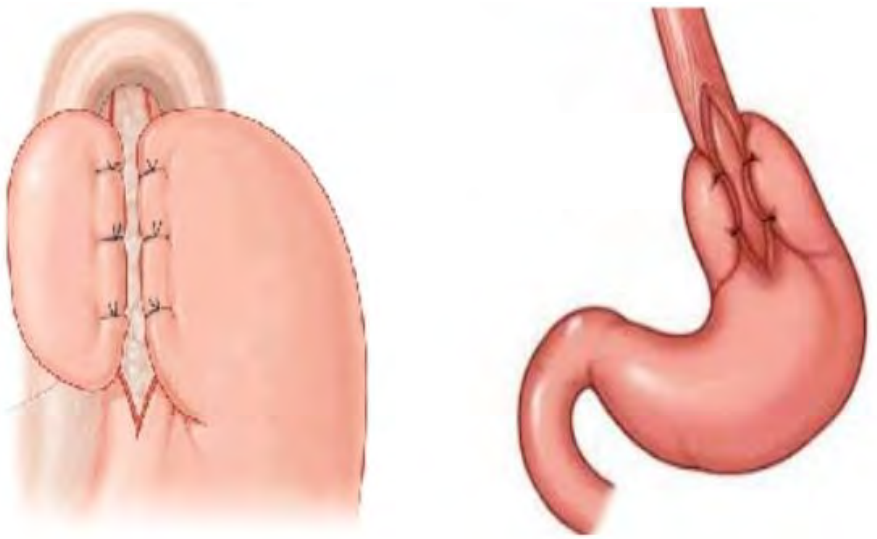
Guy E. Boeckxstaens, M.D., Ph.D., Vito Annese, M.D., Ph.D., Stanislas Bruley des Varannes, M.D., Ph.D., Stanislas Chaussade, M.D., Ph.D., Mario Costantini, M.D., Ph.D., Antonello Cuttitta, M.D., J. Ignasi Elizalde, M.D., Uberto Fumagalli, M.D., Ph.D., Marianne Gaudric, M.D., Ph.D., Wout O. Rohof, M.D., André J. Smout, M.D., Ph.D., Jan Tack, M.D., Ph.D., Aeilko H. Zwinderman, Ph.D., Giovanni Zaninotto, M.D., Ph.D., and Olivier R. Busch, M.D., Ph.D., for the European Achalasia Trial Investigators*

Boeckxstaens N Engl J Med 2011

Management Of Achalasia – Pneumatic Dilation vs. Heller's Myotomy

- Complications of treatment
  - Perforation
    - Pneumatic dilation (PD)
      - Esophageal perforation 4%
      - 3 perforations with 30mm, 1 with 35mm
      - 2 underwent surgery, 2 conservative care
    - Heller's myotomy (HM)
      - Mucosal tear in 13/106 (12%)
      - Repaired during initial surgery
  - GERD
    - Increased acid exposure similar: 15% PD, 23% HM
    - Erosive esophagitis similar: 19% PD, 21% HM
- Conclusion
  - Effectiveness of PD is comparable to laparoscopic HM if allow for repeated dilations and accept risk of esophageal perforation

Heller's Myotomy



Management Of Achalasia – Per Oral Endoscopic Myotomy (POEM)

- Peroral esophageal myotomy
  - Originally described in porcine model by Pasricha
  - First described in a patient by Inoue at DDW 2009, followed by report of 17 patients
  - Now known as per oral endoscopic myotomy (POEM)
- Clinical results of POEM for achalasia
  - 300 consecutive cases in a single site prospective study in Japan
  - 2008-2012
  - 41 cases with sigmoid esophagus, 10 prior surgical failures
  - Success (Eckardt <3) in 98%
  - 5 cases received second POEM
  - Mean operating time 110 minutes
  - 9 complications: 1 pneumothorax, 1 hematoma, 1 peritonitis, 6 mucosal injuries
  - 5% received PPI post POEM

Management Of Achalasia – Endoscopic vs. Surgical Myotomy

- Prospective multicenter study of POEM
  - Compared to retrospective cohort of lap Heller's myotomy
  - Primary outcome of symptom relief at 3 months
  - 70 patients underwent POEM
  - Mean operative time 105 minutes (54-240 min)
  - No conversions to open or lap surgery
  - Treatment success in 97% with POEM
  - POEM had significantly better 3 month symptom scores (1 vs. 1.4) and LES pressure (9 vs. 12 mmHg) compared to review of HM
  - Reflux esophagitis higher in POEM but not statistically significant (41% vs. 28%)
- Conclusions
  - Excellent outcomes of POEM are comparable to HM and reproducible in multiple centers
  - GERD complications may not be as significant as feared, perhaps due to avoidance of hiatal dissection
  - Growing experience supports effectiveness of POEM
  - POEM avoids surgical alteration of the EGJ morphology
  - POEM may become primary approach to treatment of achalasia

Secondary Achalasia

- Etiologies
  - Pseudoachalasia
  - Post fundoplication
  - Chagas' disease
  - Eosinophilic esophagitis
  - Allgrove's syndrome
  - Paraneoplastic syndrome
  - Parkinson's disease
  - MEN IIB
  - Familial achalasia
  - Sjogren's syndrome
  - Post vagotomy
  - Amyloidosis

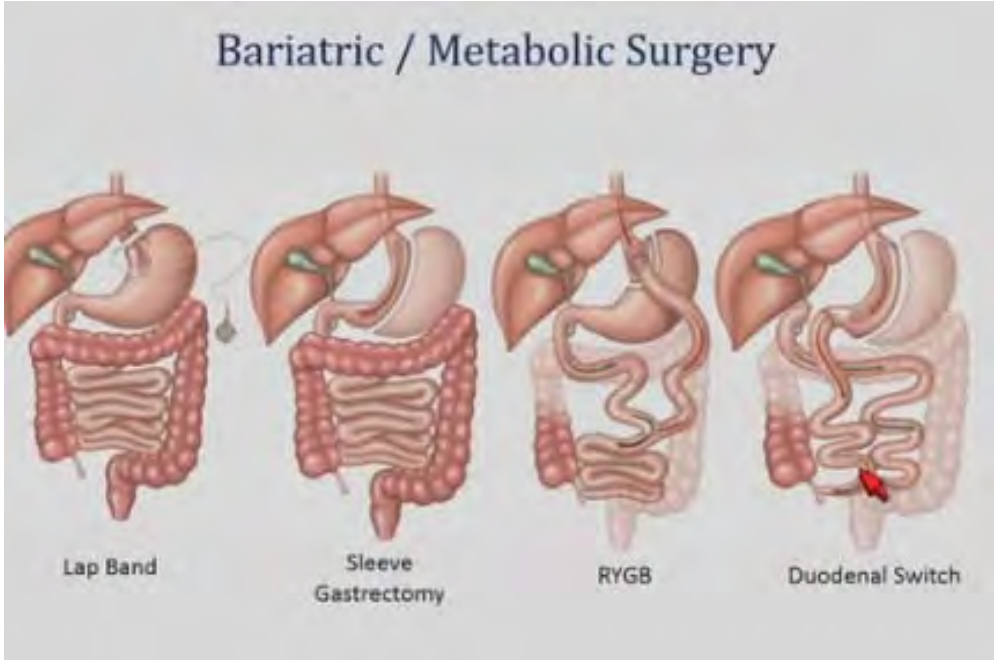
Pseudoachalasia (secondary to cancer)

Mechanical obstruction can mimic the radiographic and manometric features of gastrointestinal dysmotility

ESOPHAGEAL SYMPTOMS IN PATIENTS AFTER BARIATRIC SURGERY

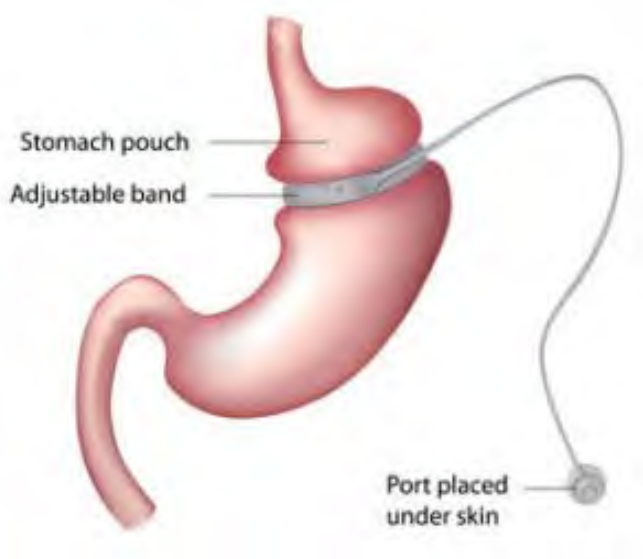
The Bariatric Patient

- Bariatric surgery can affect the LES and the esophageal body
- Laparoscopic adjustable gastric band
- Laparoscopic sleeve gastrectomy
- Roux-en-Y gastric bypass



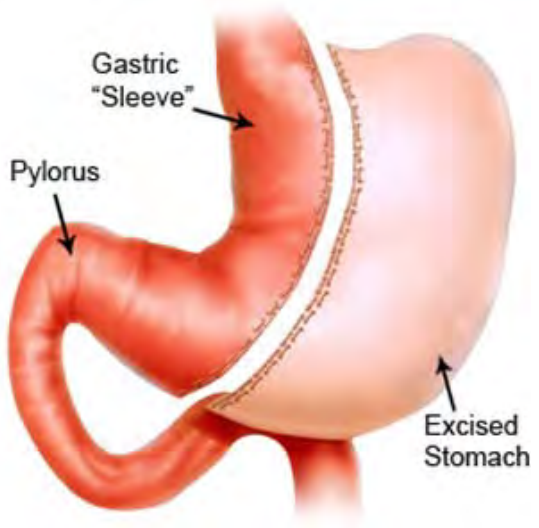
Lap Band

- Associated with:
  - weak esophageal motility
  - pouch dilatation
  - Increased LESP
- Can present as:
  - worsening GE reflux
  - esophageal stasis
  - achalasia-type symptoms



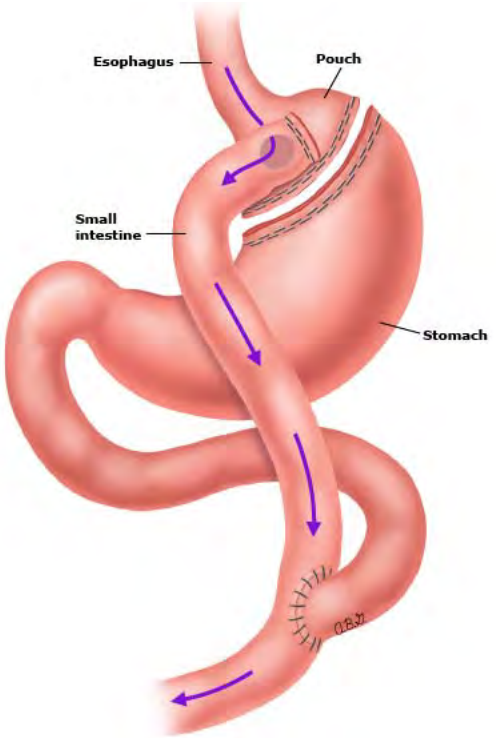
Laparoscopic Sleeve Gastrectomy

- Associated with:
  - weak LESP
  - decreased gastric compliance
  - disruption of EG junction competency
- Can present as:
  - worsening GE reflux



Roux-en-Y Gastric Bypass

- Can improve or worsen GE reflux disease
- Effect of weight loss on reduction in GERD symptoms



Live Long and Prosper

Q&A